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SCIENTIFIC PROOF IN RESPECT TO INJURIES OF THE HEART

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Does this man really have heart trouble, and if so, what kind?

Could it be caused or aggravated by an injury or by the strain of unusual effort?

If so, was there such injury or strain in this case?

And finally, was such injury or strain actually responsible for the heart trouble here, and to what degree?

In this study we shall try to answer those questions. They are being asked more and more frequently in these days of increasing interest in both heart disease and industrial medicine. Heart disease is common and "heart symptoms" and functional disorders without real heart disease are even more common. The limelight is on them and the nation has become very heart conscious. An immature publicity is in part at least responsible for an unreasoned dread of anything that suggests heart trouble. An important function of the medical profession in our generation is to dispel, with confidence based on knowledge, the exaggerated concern about heart disease so widely spread abroad; the legal profession can help physicians to fulfill this obligation.

It will be well for us first briefly to outline the more important anatomical and physiological features of the circulation, ignorance concerning which is responsible for many misconceptions, in court and out, and to recount the various methods of cardiovascular examination and

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their relative value. Then we shall consider the various types of heart disease and their relation to injury and strain before discussing, finally, pervasive medicolegal aspects of cardiac conditions.

Anatomical and Physiological Considerations

The Heart a Muscular Pump. The circulation of blood is carried on by the help of several different structures and mechanisms of which the heart is only one though by far the most important one. The very first and essential comment about the heart itself is that it is composed of tough and powerful muscle, called the myocardium, so arranged about the central reservoirs of blood that it acts during systole (contraction) as a force pump, with some minor suction action also during early diastole (relaxation); it is not easily injured and when it is, it possesses an unusual recuperative power. Now as to its parts.

The Auricles, the Receiving Chambers of the Heart. The right and left auricles of the heart (Figure 1) receive the blood from the great veins of the systemic circulation (superior and inferior venae cavae) and of the pulmonary circulation (right and left pulmonary veins) respectively, and being thin walled and distensible are able to hold a variable but fairly large volume, ordinarily from 60 to 150 c. c. (2 to 5 ounces) each. From these two receiving chambers the blood flows during the diastole of the heart into the ventricles through the right and left auriculoventricular valves which are called the *tricuspid* and *mitral* valves respectively.

The Pacemaker of the Heart and the Spread of the Electrical Stimulus through the Heart. The Electrocardiogram. At the end of diastole, which constitutes the pause in the heart's action between beats, a minute electric current is generated, normally 50 to 100 times a minute, in a tiny special structure at the junction of the superior vena cava and right auricle called the sinoauricular node or pacemaker. This electrical impulse travels from its point of origin as a wide wave over the walls of both auricles causing or accompanying the contraction of the muscle fibers in those walls. This process gives rise to a special deflection called the *P* wave in the electrical tracing of the heart beat (electrocardiogram) and to a movement of the pressure curve in the heart itself due to the contraction of the auricular chambers (Figure 2). The auricular contraction or systole squeezes the last of the blood as it flows into the ventricles at the end of the diastole of the latter, but its more important function is to set off in its turn the contraction of the ventricles via the so-called excitation wave which is represented electrically by the *P* wave. In the junctional tissue between auricles and ventricles, consisting for the major part of fibrous connective tissue with

blood vessels (coronary) and nerves embedded in it, there lie special structures which carry the excitation wave from auricles to all parts of the ventricular muscle. These special structures are called the a-v (auriculoventricular) node (of Tawara), the a-v bundle (of His), and the bundle branches right and left, and distal fibers (of Purkinje). The systole of the auricles lasts from 0.1 to 0.2 second. The time interval between the beginning of the auricular wave and the beginning of the first ventricular wave (the QRS of the electrocardiogram) is called the P-R interval and normally it is between 0.1 and 0.2 second.

The Ventricles and Their Systole. The Valves and the Heart Sounds. The right and left ventricles of the heart are pumping chambers with thick muscular walls and are separated by a muscular septum. Soon after birth the left ventricle becomes much more powerful than the right ventricle and eventually it develops a wall three times as thick (10 mm. compared to 3 mm. in the adult). This is because the left ventricle has more work to do than the right: the left ventricle has to pump blood to many and distant parts of the body whereas the right ventricle merely pumps blood from the heart to the lungs for oxygenation. The ventricles contract simultaneously. As soon as the pressure in their chambers rises sufficiently, which is early in their systole (contraction) the tricuspid and mitral valves snap shut giving rise to the *first sound* of the heart (Figure 2). This sound can be heard with the stethoscope or, indeed, with the naked ear applied to the front wall of the thorax (the chest), particularly over the area where the lowest part of the heart, the apex, thrusts against the chest wall (the region of the cardiac apex impulse). In addition to the tricuspid and mitral valves, the heart has two further valves: the pulmonary valve and the aortic valve. The pulmonary valve separates the right ventricular cavity from the main pulmonary artery. The aortic valve separates the left ventricular cavity from the aorta, the large artery which conveys freshly oxygenated blood from the heart to the body. Soon after the first heart sound is heard, the pulmonary and aortic valves both open widely thus permitting blood to flow from the heart into the great arteries. The blood which flows through the open pulmonary valve into the pulmonary artery is carried to the lungs where its lost oxygen is restored before it returns, via the pulmonary veins, to the left side of the heart. The blood which passes through the open aortic valve into the aorta and thence to the body tissues, is freshly oxygenated blood which the heart has just received back from the lungs.

Systole. The beginning of ventricular systole (contraction) is represented in the electrocardiogram (the electrical tracing of the heart beat) by the so-called QRS wave and by a sharp rise in the pressure

curve. Ventricular systole lasts between 0.25 and 0.50 second, depending mainly on the heart rate: the slower the rate, the longer the systole. The end of ventricular systole is represented by the end of the *T* wave in the electrocardiogram, a drop in the intraventricular pressure curve, and the second sound of the heart. This second heart sound results from the closure of the pulmonary and aortic valves. Usually the valves close simultaneously, giving rise to a single sound. Sometimes, even in normal persons, the two valves do not close at the same instant, and this results in what we call a "split" second sound. (In a similar manner the first heart sound may be "split" if the tricuspid and mitral valves do not close synchronously.) During systole of the ventricles the *T* wave is written by the electrocardiogram. The *T* wave indicates two things: the "retreat" or subsidence of the electrical excitation wave and the state of the myocardium, or heart muscle. It is one of the most delicate evidences we possess of the condition of the heart muscle (myocardium) and the *T* wave may be much distorted when there is no other sign of trouble.

Diastole. Diastole represents a period of rest for the heart muscle (myocardium). During this time there is a building up of electrical energy in the sinoauricular node preparatory to its next discharge. The ventricular muscle becomes relaxed, the ventricular chambers dilate, and the tricuspid and mitral valves then open. The valves open partly as a result of dilatation of the ventricular chambers with ensuing negative pressure therein and partly as a result of the positive pressure in the auricular chambers. This positive pressure results from the fact that during their own earlier diastole the auricular chambers have been filling up with venous blood returned to the heart by way of the great veins. When these two valves open, blood pours down into the ventricles from the auricles and if there is a fairly long interval between heart beats, say 0.6 second, the ventricular chambers become quite full before the next impulse starts in the right auricle.

Work of the Heart. At average heart rates the heart muscle has about an eight hour day of work but it has to snatch its rest periodically every second. With a pulse rate of 60 (per minute), systole lasts about 0.35 second and diastole 0.65 second. With a pulse rate of 120 systole is shortened to 0.25 second and diastole much more so, both relatively and absolutely, to 0.25 second. With the left ventricle pumping 100 c. c. of blood per beat at a rate of 60 beats per minute, and at a mean arterial pressure of 100 millimeters of mercury, it actually does the work of lifting six liters (about six quarts) of blood to a height of 10 centimeters (approximately 4 inches) of mercury (or about 130 cen-

timeters of its own weight) per minute. This is the equivalent of lifting nearly 20 pounds a yard into the air every minute.

Other Parts of the Structure of the Heart Besides the Muscle and the Valves. Besides the parts of the heart mentioned above, attention may well be called to the *pericardial sac* in which the heart lies. This important sac has the dual function of lubricating and protecting the heart. The pericardial sac has an outer (parietal) layer and an inner (visceral) layer; these two parts of the so-called *pericardium* invest the heart and protect it from direct contact with the lungs and pleural cavities on both sides and from other structures in the chest. The inside of the heart is lined by a delicate tissue called the *endocardium* which covers the inner fibrous valves as well. Finally, nerves and blood vessels (coronary arteries and veins) course into the walls of the four heart chambers from main sources of supply outside. The function of the nerves is to help slow or speed the heart or otherwise regulate its action according to bodily demands. An organ which works so continuously as the heart needs a rich blood supply to bring oxygen and food and to carry away waste products. The coronary circulation serves the heart exclusively, carrying to the heart muscle almost one-fifth of the freshly oxygenated blood pumped by the left ventricle. One can understand that interference with the heart's essential blood supply, by narrowing or obstruction of the coronary arteries, always involves serious risks of injury. We shall have much more to say about the coronary arteries as our discussion progresses.

The Size and Position of the Heart. The Wide Range of the Normal. The normal *size* and *position* of the heart are of great importance as a basis for determining when abnormal enlargement and displacement have been caused by disease. Unfortunately, however, the size and position of the heart are extremely variable from one person to another as a normal finding and even in the same person position of the heart may vary from time to time. The range of the normal heart as recognized at present, based on inadequate criteria, is so great that only the most careful serial studies of an individual as he passes from health into sickness can answer certain questions concerning these points. Such studies are very rare, especially when any particular case comes up for consideration. One can merely say that the weight of the normal adult heart varies from 200 to 400 grams, depending to a considerable degree on the size of the individual; a very rough average is a ratio of heart weight to body weight of 1 to 200. Here, however, build is probably more important than weight but to date this relationship has been inadequately explored. The volume of the heart tissue itself ranges around 300 c. c. With the chambers filled, the volume of

the entire heart comes to about 750 c. c. or ¾ of one liter. The contour of the heart is more or less of an inverted egg shape with the great blood vessels acting as a pedicle above from which the heart hangs in the pericardial sac with apex and inferior surface supported below by the diaphragm, the circular, flat muscle which divides the chest cavity (thorax) from the abdomen. The position of the heart in the thorax is essentially median but with the apex and the greater bulk of the heart more to the left than to the right in the great majority of cases (Figure 3). It is thus a mediastinal structure[1] like the trachea (wind-pipe), the esophagus (gullet), and the aorta (main artery from the heart) which lie behind and above it, flanked by the two lungs. The anterior chest wall (sternum, ribs, and muscles) lies directly in front of the heart, and behind it, beyond the trachea and esophagus, is the spinal column. If the thorax (chest cavity) is very shallow the heart may be compressed anteroposteriorly, but usually it is merely displaced to the left, as it tends to be transiently, without serious injury if the chest itself is acutely compressed. If the thorax (chest cavity) is short and the diaphragm high, as in many obese persons, the heart is pushed up, lies in a transverse position and looks larger than it actually is.

Other Aids to the Circulation of Blood. The Aorta. So much, in brief, for the heart but we would be guilty of a serious error of omission if we said nothing about the other structures that help to maintain an adequate circulation. First, the great arteries, in particular the aorta, have an important function, not merely in acting as tubes to convey blood, but by their elasticity in helping to maintain a constant flow of blood. Thus, when the left ventricle ejects its content of blood into the aorta during systole, the stream not only moves forward during that period of time but distends the very elastic aortic wall which thus takes up some of the force of the cardiac contraction. During the diastole (resting phase) of the left ventricle, the aorta returns to its usual resting caliber. It exerts pressure in doing so which serves to close the aortic valve and also to propel the blood more evenly towards the periphery of the body and backward into the myocardium[2] itself via the network of the two coronary arteries which arise from the very first portion of the aorta. With age, and sometimes prematurely, the aorta loses its elasticity and hence no longer aids the circulation of blood.

Peripheral Arteries. As one approaches the peripheral circulation one finds more muscle and less elastic tissue in the walls of arteries.

[1] *Mediastinum:* The mediastinum is the space in the middle of the thorax which lies between the lungs on each side and between the sternum (breast bone) in front and the spine in back.

[2] *Myocardium:* The myocardium, or heart muscle, is specialized striated muscular tissue with branching cells forming thick muscle masses in the ventricles and thin layers of muscle in the auricles.

This permits a localization of compressing action on the blood stream and thus a regulation of the blood volume which reaches a part. Larger arteries subdivide into smaller ones until finally one comes to the smallest arteries of all, the arterioles. These small arteries have the remarkable ability to close entirely for various periods of time, thus shunting the blood from parts of the body where it is not then needed to parts where it is (as from muscles to the gastrointestinal tract and *vice versa*).

Capillaries. Next we reach the capillaries where the chief purpose of the circulation is achieved, namely, the two-way diffusion of chemicals and gases between the blood and the body cells. This is possible because the tiny thinwalled capillary vessels permit an interchange, by means of hydrostatic[3] and osmotic pressure,[4] of chemicals and gases between the blood and the body cells, the direction depending upon the pressure relations temporarily existing between the two. In general, hydrostatic pressure is higher near the beginning of a capillary, causing diffusion from blood to tissue; near the end of the capillary hydrostatic pressure tends to be low and osmotic pressure causes ingredients of the tissue fluids to diffuse back into the blood.

The Veins and Their Valves. The veins collect the blood from the capillaries and transport it back to the heart. This process is promoted by the fact that the veins contain valves, structures whose true significance was recognized by Harvey as a fundamental clue in his discovery of the circulation early in the seventeenth century. The valves prevent the backward flow of the blood while three positive forces produce the forward flow on toward the heart. One of the three forces mentioned is the hydrostatic pressure which started at a high value in the aorta, decreased to what we know arbitrarily as "the blood pressure" in the middle sized vessels as in the arms, dwindled to a relatively low pressure in the arterioles and capillaries but was still positive in the small veins.

The Tone of the Skeletal Muscles. The Diaphragm. The second force is the compression of veins by the skeletal muscles, both in contraction and in relaxation. The third force is the contraction of the diaphragm (the circular sheet-like muscle separating the chest cavity and the abdomen) which acts like a piston in a large caliber pump sucking not only air into the lungs but blood into the great veins and thence to the heart.

The Heart Rate. Only two more points of practical importance remain in this introductory section concerning the anatomy and phys-

[3] *Hydrostatic pressure:* Hydrostatic refers to pressure mechanically exerted by a fluid in a state of rest or equilibrium.

[4] *Osmotic pressure:* Osmosis is the passage of pure solvent from the lesser to the greater concentration when two solutions are separated by a membrane which selectively prevents the passage of solute molecules, but is permeable to the solvent.

iology of the circulation. The normal heart rate is often put down as 72 per minute but here especially there is a tremendous range, from 35 in a well trained athletic champion at rest to 200 in a poorly trained man during very vigorous effort. Even a well trained athlete, however, may as a result of nervousness, show a pulse rate of over 100 at rest. If the heart and circulation are healthy they can easily stand all these variations of rate with ease.

The Blood Pressure. The other point concerns the blood pressure. As ordinarily taken, over the brachial artery at the elbow, the arterial pressure shows a normal range during systole between 90 and 150 mm. mercury, while the diastolic pressure, which is much more important because it is a constant baseline tension, ranges from 60 to 90 mm. The pressure in the aorta is of course much higher, in the larger leg arteries also higher, in the fingers considerably lower, and very low by the time the finest blood vessels are reached. Finally, the pressure in the veins can be measured, too, best by direct readings, where necessary; in an average arm vein it ranges normally from 3 to 8 centimeters of blood or water but varies considerably depending on the technique employed.

Methods of Examination

The Patient's History. By far the most important method of cardiovascular examination is that of the taking of the history, present and past. This involves obtaining a reliable account of the patient's previous illnesses, especially rheumatic fever, syphilis, and nervous prostration or nervousness pure and simple, operations, and accidents. One is also interested in learning what previous physical examinations (for insurance, military service, or routine health appraisals) revealed concerning heart size, rhythm, and rate, murmurs, and blood pressure. Heart and nervous troubles in the family may afford clues since some cardiovascular complaints have an hereditary tendency; this is true especially of rheumatic heart disease, high blood pressure, coronary heart disease, and nervous heart trouble. The individual's habits as to exercise, tobacco, alcohol, and intensive nervous strain in work or otherwise may be significant. And finally, the symptoms need careful analysis; there may be none in the face of important heart disease or there may be many with no heart disease at all. Experience in eliciting and analyzing the story may be worth much more than all the objective evidence in the world—in fact it counts for at least half of the total value of the complete examination in the average case.

Cardiac Symptoms. There are only two important cardiac symptoms: *angina pectoris* (suddenly recurring severe pain in the chest) or

the similar pain of acute myocardial infarction;[5] and, secondly, *dyspnea* (i.e., breathlessness) when it is due to disease of the heart. Sometimes these are obvious on observation but often they must be elicited by questioning or by testing. Angina pectoris and the pain of acute heart muscle infarction are oppressive in nature, are usually severe, and appear under the breastbone in position with, or without, radiation to the arms. Characteristically, angina pectoris lasts only a few minutes, comes on exertion, and is relieved by rest or nitroglycerine while the pain of acute cardiac infarction usually lasts hours at a time, generally occurs while the patient is at rest and requires morphine for its relief. This pain must be differentiated from that due to spasm of the esophagus or stomach and from gall bladder disease; it is the result of aching of heart muscle inadequately supplied with oxygen. Breathlessness due to heart failure occurs earliest on effort but may later come at rest. Such breathlessness is due to congestion of the lungs with blood, which does not allow enough air to enter the bronchioles and alveoli to permit a proper exchange of oxygen and carbon dioxide; *it is always associated with enlargement of the heart which is failing* and must be differentiated from shortage of breath due to disease of the lungs or other causes.

Physical Examination. In determining a person's cardiovascular condition, a carefully taken medical history is of first importance to the physician, but physical examination is a close second, rating ahead of such special methods of study as electrocardiography. The various forms of heart disease tend to produce distinctive alterations in the heart itself (characteristic types of enlargement, shifts in position; change in particular heart sounds caused by lesions of particular valves; changes in heart rate or rhythm, etc.) or in other parts of the body, these latter being as a rule associated with the partial failure of the heart as a pump. The examining physician knows that if he can make enough vital observations, he can determine whether the heart is sound or impaired, and the nature of the disorder.

How then, does the physician proceed in acquiring these data? First he will learn what he can about the heart itself by inspection, palpation, percussion and especially auscultation. Let us note briefly what each of these diagnostic steps entails:

(1) *Inspection:* Inspection consists of critical visual examination of the body, front and back, in a search for abnormalities of pulsation of

[5] *Infarction:* Infarction involves damage consisting of a variable degree of destruction of tissue in any organ or structure of the body due to cutting off of its blood supply. When it involves the heart muscle, the condition is called myocardial infarction.

heart or arteries, of shape of the thorax itself, of excursion of respiration, of abnormal color, and of prominence of arteries and veins.

(2) *Palpation:* Palpation is the method of examination by feeling the thorax with the palm of the hand and fingers in a search for position of the cardiac apex impulse, thrills produced by unusual currents of blood in the heart or great vessels, and unusual pulsations over the region of the heart or elsewhere in the chest.

(3) *Percussion:* Percussion consists of a method of examination by tapping with the fingers on the chest to determine the varying degrees of resonance and dulness. The heart itself presents an area of relative dulness to the percussion finger while normally there is a well marked resonance over the lungs. If the heart is much enlarged, there tends to be an increase in percussion dulness and the same thing is true when there is fluid in the pleural cavities or in the pericardium.

(4) *Auscultation:* Auscultation consists of a method of examination by listening to the heart and lungs with a stethoscope over the chest. By listening over the heart itself, one analyzes the heart sounds and notes the presence or absence of murmurs which are normally or abnormally produced by currents of blood. Auscultation over the lungs determines the character of the breath sounds and the presence or absence of râles, that is, bubbles, or of wheezing.

As we have said, the doctor hopes to gain invaluable clues, also, from the results of his general examination. This includes consideration of the person's appearance, appraisal of his or her nervous state and intelligence and of the probable accuracy of the history related, and the mental attitude of the individual towards any trouble he may have. In addition, the doctor is alert to discover those little indications here and there about the body which constitute eloquent language to the heart-specialist, proclaiming as they do the degree to which the cardiovascular system is measuring up to its full-time job or is falling short. Among these evidentiary "signposts" which help guide the examining physician are these:

(1) *The patient's color:* is it normal or bluish (cyanotic)?

a. *Cyanosis* is a blue color of varying degree, most prominent when present in the lips, tongue, cheeks, ears, and finger tips. It is due most commonly to disease of the lungs but may be the result of certain heart troubles, in particular congestion of the lungs resulting from failure of the left ventricle. and congenital heart disease which consists of deformities of the heart present from birth by which venous or blue blood is shunted into the general or systemic circulation.

(2) *The patient's thyroid gland: is it functioning normally or ab-*

normally? A hyperactive thyroid gland may cause rapid heart action and palpitation.[6]

(3) *Does the patient have an abnormal pulse in his neck?* If so, this may be due to:

a. *Aortic regurgitation:* which consists of leaking of blood backward from the aorta into the heart due to defective closure of the aortic valve; or

b. *Hypertension:* which consists of an abnormal elevation of the blood pressure (in the adult above 150 millimeters of mercury systolic or above 90 diastolic pressure); or

c. *Aneurysm,*[7] if the carotid artery[8] in the neck is much dilated or pulsates overactively; or

d. *Congestion* from failure of the heart muscle—if the jugular veins[9] are overfull or pulsate when the subject is in an upright position.

(4) *Does the patient have râles,*[10] *or consolidation of the lungs,*[11] *or fluid in the pleural spaces* (i.e., the space between the two layers of

[6] *Thyroid gland:* The thyroid gland is a gland in the front of the neck over the larynx and trachea which functions normally to maintain an adequate metabolic rate, that is, exchange of gases and chemicals throughout the body. Overaction of the thyroid gland increases the metabolic rate and causes a disease called hyperthyroidism or thyrotoxicosis while a decrease in the activity of the thyroid gland produces a sluggish action of the body called myxedema in adults or cretinism in children.

Palpitation: Unduly rapid, irregular or forceful action of the heart which is felt by the patient.

[7] *Aneurysm:* A sac formed by the dilatation or ballooning out of a weakened portion of the wall of an artery or of the heart. It is filled with blood. The chief symptoms of aneurysm are the formation of a pulsating tumor (swelling), a peculiar bruit (abnormal sound) heard over the swelling when one listens with a stethoscope, and pressure symptoms, consisting of pain and paralysis from pressure on nerves and absorption of contiguous parts.

An aneurysm of the thoracic aorta is due almost invariably to syphilis, of the abdominal aorta to arteriosclerosis, and of the peripheral arteries to trauma or injury. In the case of the heart an aneurysm is usually due to weakening of the wall from *myocardial infarction* (deprivation of blood supply to the heart by clotting of blood in one of the coronary arteries with resultant coronary occlusion or thrombosis).

[8] *Carotid artery:* The principal artery of the neck, one on each side: it arises from the large arteries near the heart (the innominate on the right side; the arch of the aorta on the left side) and divides into the external and internal carotid arteries which carry blood to the face, ears, neck, head, skull and brain.

[9] *Jugular veins:* The jugular veins are the veins in the neck, one on each side, which bring venous blood (low in oxygen) back from the head to the innominate veins which in turn empty into the superior vena cava and the right auricle; the blood passes thence to the right ventricle which pumps it to the lungs for restoration of its oxygen. There are two sets of jugular veins on each side, the external and the internal jugular veins.

[10] *Râle* (rahl) (Fr. meaning "rattle"): Any abnormal respiratory sound heard when one listens to the patient's breathing through a stethoscope applied to various portions of the chest wall overlying the lungs (auscultation). Their presence indicates an abnormal condition possibly due to disease. For diagnostic purposes râles are classified according to their sound qualities and location.

[11] *Consolidation of the lungs:* Solidification of the lung tissue due to an inflammatory reaction resulting from pneumonia or hemorrhage, or from an infarction (note 5, *supra*) caused by embolism to the lung (note 12, *infra*).

the pleural sac which invests each lung)? If so, this may be due to heart failure or to pulmonary embolism.[12]

(5) *Does the patient have enlargement of the liver and fluid in his peritoneal (abdominal) cavity?* If so, this indicates that there is much congestion due to heart disease (this cause for enlargement of the liver is to be differentiated from cirrhosis of the liver).[13]

(6) *Does the patient have swelling (edema) or faulty arterial pulse or blocked veins in the legs?* If so, this indicates interference with the peripheral circulation due to a central cause (failing heart) or due to a peripheral cause (defective veins).

The estimation of the blood pressure forms a part of the routine cardiovascular examination and if the subject's pressure is a little elevated from nervousness at first, the determination may need to be repeated after he has rested for half an hour in a recumbent position.

Physical Examination of the Heart. The cardiac examination itself is important for four particular reasons:

(1) To determine heart size by the position of the apex impulse and left border of percussion dulness when careful X-ray study is not possible. (The apex impulse and left border of percussion dulness should not be beyond, that is, to the left of, the midclavicular line[14] in the fifth intercostal space.);[15]

(2) To note the character of the heart sounds by stethoscope[16] which may be more significant than the presence or absence of murmurs;

[12] *Pulmonary embolism:* Pulmonary embolism consists of a blocking of one or more arteries in the lungs by blood clots brought there in the blood stream. These clots come through the right heart and the veins, usually from some distant part of the body as in the case of phlebitis (inflammation) in the leg veins, but they may rarely arise from the right heart chambers themselves. Usually pulmonary embolism produces pulmonary infarction—i.e., injury and even death of a certain amount of the lung tissue dependent upon the blocked arteries for their blood supply.

[13] *Cirrhosis of the liver:* A liver disease often due to chronic alcoholism, but sometimes to other causes, involving an initial enlargement of the liver and replacement of normal tissue with fibrous elements; these contract with passage of time causing shrinkage and hardening of the liver.

[14] *Midclavicular line:* The midclavicular line is a line drawn vertically from a point midway between the central vertical line of the chest and the place near the point of the shoulder where the clavicle (collarbone) and the scapula (flat bone of the shoulder) come into apposition to form a joint. This line is an important landmark for determining the heart size on physical examination; the apex impulse and left border of the heart should not lie beyond, that is, to the left of this line.

[15] *Fifth intercostal space:* The left fifth intercostal space is the interval between the fifth and sixth ribs on the left side and is an important landmark for the location of the impulse of the heart, which normally lies therein. If the apex impulse lies below the fifth intercostal space, this almost always indicates enlargement of the heart.

[16] *Stethoscope:* The stethoscope is an instrument used for auscultation, that is, for listening to the heart and lungs. It may be connected with the ears directly or with electrical apparatus, microphone and galvanometer so as to permit graphic recording of heart sounds and murmurs.

(3) To listen for important heart murmurs which point either to valvular defects or to dilatation of the heart or aorta; and

(4) To detect arrhythmias,[17] better revealed, however, by electrocardiography as this provides a tracing of the heart beat during a given period of time.

Thus, the physical examination requires careful scrutiny of all parts of the body and under certain circumstances it should be still more complete; for example, if high blood pressure is present or is suspected to have existed in the past the eye grounds[18] should be inspected by ophthalmoscope[19] to see how much sclerotic change[20] is present in the small arteries therein.

Relative Values of the Methods of Examination. If we grade the value of the history as 50 per cent and that of the physical examination as 30 per cent, the electrocardiogram would rank about 10 per cent, X-ray examination 5 per cent, and all other tests the remaining 5 per cent.

Electrocardiography consists of the study of the electrical manifestation of the heart's action. Developed in the physiological laboratories it has become an important tool in clinical medicine not only in the diagnosis of certain cardiac conditions but in follow-up study of certain patients. The electrocardiogram consist of the record taken by the electrocardiograph, an instrument consisting primarily of an electromagnet with recording string or mirror whose shadow or beam of light is projected on a moving photographic film. Every time the heart beats there are minute currents which produce certain waves or deflections described in the early part of this article. Distortion of these waves (P associated with the contraction of the auricle and QRS and T associated with ventricular contraction) may be produced by toxic influences or disease processes.

X-ray examination of the heart consists of recording or observing the shadows of the heart and great vessels on film or screen (by fluoroscope[21]). X-ray study gives by far the most accurate idea of heart

[17] *Arrhythmias:* Arrhythmias consist of irregularities or other disturbances of rhythm of the heart beat.

[18] *Eye grounds:* The eye grounds are the retinae containing the visual end organs of the eyes; they are inspected with an ophthalmoscope in order, particularly, to study the condition of the small blood vessels there.

[19] *Ophthalmoscope:* The ophthalmoscope is the instrument used for inspecting the eye grounds. It has a source of illumination and lens for magnification.

[20] *Sclerotic change:* Sclerotic change consists of the hardening and usually increase in length (with tortuosity) of arteries anywhere in the body. It is often an associated finding accompanying high blood pressure.

[21] *Fluoroscope:* The fluoroscope is a device used for examining deep structures by means of X-rays: it consists of a screen (fluorescent screen) covered with crystals of calcium tungstate. A great advantage of this method of examination over that of the study of the X-ray film is that in addition to size and shape of

size and shape and provides the only feasible means of examining the left auricle, the blood vessels of the lungs, and much of the aorta.

We would rank electrocardiography as more important than X-ray examination of the heart because it may reveal abnormalities of the heart muscle which are to be found in no other way, in particular coronary or traumatic heart disease, while the X-ray film or fluroscopy usually does no more than to confirm the impression already gained by the other methods of study. No cardiovascular examination is complete without both electrocardiogram and X-ray study, and in certain persons of unusual build or with pulmonary emphysema[22] the only way to determine the very important measurement of heart size is by the X-ray.

Other Methods of Examination. Routine blood and urine examinations should be done to rule out rare causes of cardiovascular abnormalities, and infrequently special tests to determine the speed of blood flow, venous pressure, vital capacity,[23] and the effect of exercise during low oxygen inhalation may yield information needed to complete the analysis of a case. Finally, neuropsychiatric consultations may be crucial in determining to what degree the patient's symptoms are due to nervous or mental states and to what degree they are caused by abnormalities of the heart or circulation.

Proof of the Presence of Heart Disease and of Its Various Types and Their Prognosis

The heart is abnormal if any one or more of several conditions exist:

(1) *If the heart is enlarged.*[24]

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the heart and of the great vessels, it is possible to observe the force and extent of the contraction of the heart and the movement of the leaves of the diaphragm, the large circular muscle which separates the chest cavity (thorax) and the abdominal cavity.

[22] *Pulmonary emphysema:* Pulmonary emphysema consists of the excessive distension of the air sacs and passages in the lungs with air, as in asthma. This decreases the efficiency of the lungs.

[23] *Vital capacity:* The vital capacity consists of the fullest amount of air, measured usually in liters, that can be exhaled in one expiration following a maximum inspiration. It is determined by having the patient inhale deeply and then blow his breath into a *spirometer,* an instrument which records the volume of air so exhaled.

[24] *Cardiac enlargement:* Enlargement of the heart of moderate or advanced degree is readily recognizable by X-ray study and also as a rule by physical examination. But if the cardiac enlargement is slight it may not be ascertainable by any method, except serial (for example, annual) X-ray measurements of heart size and shape in the same individual carefully made before and after the occurrence of some factor of strain. As it is now, the most satisfactory standards, far from adequate, involve the Hodges-Eyster nomogram (a chart or diagram on which a number of variables are plotted, forming a computation chart for the solution of complex numerical formulae). This is based on height, weight, and age, and the transverse diameter and area of the heart shadow. However, even using such standards, it is possible for the cardiac area to increase 75 per cent

(2) *If the heart presents defective sounds* (such as marked accentuation of either sound, very weak first sound at the apex in contrast to the second, absent aortic second sound, or gallop rhythm[25]—in particular, early diastolic gallop rhythm at the apex).

(3) *If there are important murmurs:* (These include more than slight[26] systolic murmurs[27] heard at the apex, at the aortic valve area, and at the left border of the sternum. At the pulmonary valve area a systolic murmur may normally be slight to moderate (grades 1, 2, or 3) but a really loud murmur (grade 4 or 5) indicates the presence of heart trouble. Lastly, all diastolic murmurs[28] must be considered as abnormal.)

(4) *If the heart shows a gross arrhythmia in its beat:* (Such as occurs in auricular fibrillation[29] or paroxysmal tachycardia[30] or heart block,[31] either auriculoventricular[32] or of bundle branch type[33] (shown only by electrocardiogram).

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and still remain within the normal upper limit. A better standard than any yet introduced is likely to include anthropometric data more dependent on build. In some persons with wide chests and relatively low heights and weights the heart size can be measured better by the old cardiothoracic ratio (normally, the ratio of the transverse diameter of the heart to the inner transverse diameter of the thorax should not be over 50 per cent). As the situation stands now we cannot satisfactorily detect slight cardiac enlargement.

[25] *Gallop rhythm:* Gallop rhythm is an unusual rhythm of the heart sounds in which a loud third sound is heard and the total sound effect suggests the gallop of a horse.

[26] *Grading heart murmurs:* It is convenient to use Levine's classification of intensity of heart murmurs: grade 1 = very slight, grade 2 = slight, grade 3 = moderate, grade 4 = loud, and grade 5 = very loud.

[27] *Systolic murmur:* A systolic murmur is a murmuring sound which may be either blowing, musical, or rough produced in the heart during its systole, therefore, occurring between the first and second sounds.

[28] *Diastolic murmur:* A diastolic murmur is a murmuring sound, blowing, musical, or rough in character produced in the heart during diastole, that is, between the second and the first sounds.

[29] *Auricular fibrillation:* Auricular fibrillation consists of a gross disturbance of the heart rhythm due to the absence of rhythmic contractions of the heart. There is an incoordinate rapid action of the auricles resulting from the circuit of the excitation wave at a very rapid rate, some 300 to 400 per minute, in the neighborhood of but not in the sinoauricular node and pacemaker. It is most commonly found in rheumatic heart disease with mitral stenosis, in over-action of the thyroid gland, and in a few other conditions, even infrequently in normal persons, as from the effect of excessive tobacco.

[30] *Paroxysmal tachycardia:* Paroxysmal tachycardia consists of a spell or spells of rapid racing of the heart, regular in rhythm, at rates usually about 150 per minute, due to abnormal stimulus production somewhere in the auricular or ventricular muscle outside of the pacemaker. It is common in persons with otherwise normal hearts as well as in individuals with heart disease.

[31] *Heart-block:* Heart-block consists of a delay in conduction of the electrical stimulus of the heart as it spreads from the pacemaker down through the auriculoventricular node and bundle. There are all grades of heart-block from a slight delay which would increase the P-R interval of the electrocardiogram from .15 up to .25 of a second, to complete blocking of the impulse so that the auricles and ventricles beat independently, the former at their usual rate and the latter at a very slow rate.

[32] *Auriculoventricular block:* Auriculoventricular block refers to heart block due to a defect in the conduction of the impulse through the auriculoventricular node and bundle.

[33] *Bundle branch block:* Bundle branch block refers to defective conduction

(5) *If there is electrocardiographic evidence of myocardial disease:* (Shown particularly by flattening and inversion of the *T* waves) ;[34] and

(6) *If there is congestion of blood in the lungs, liver, or systemic veins due to failure of the heart muscle as a pump.*

Not all these findings are permanent although most of them are. Not infrequently there may be temporary enlargement of the heart, or murmurs (particularly systolic ones at the apex), or arrhythmia, or electrocardiographic changes, or even myocardial failure due to some remediable or transient condition, especially anemia,[35] excessive paroxysmal tachycardia,[35a] myxedema,[36] avitaminosis,[37] and drugs or poisons.[38] Nevertheless such conditions should be labeled heart disease or at least heart trouble, until it clears, no matter what the cause may be.

The Major Types of Heart Disease and Their Medicolegal Aspects Briefly Considered

There are three chief kinds of heart disease, namely: rheumatic, hypertensive and coronary. Less common than these are the following types of heart disease: congenital, syphilitic, bacterial endocarditis, thyrotoxic, and pulmonary. Lastly are the rare types of heart disease, namely: neoplastic (cancer), traumatic (due to injury), toxic, nutritional, and the results of uncommon infestations. Rather than overburden this manuscript with minutiae of little value to the lawyer, we shall confine our descriptions to terse indications of the main medical and medicolegal implications of each.

1.a. *Rheumatic heart disease.* Rheumatic heart disease is the most frequent type in New England but is much less common in the South. It is of two kinds: *acute and chronic;* sometimes they are combined.

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through one or the other of the two bundle branches, going either to the right or to the left ventricle. This type of heart block is almost always due to disease of the coronary arteries.

[34] *T wave:* *T* wave of the electrocardiogram is the second deflection during systole of the ventricles, the first deflection being the QRS wave; it probably represents retreat of the electrical activity from the muscle of the ventricles. It is a very important wave for study in appraising the condition of the main body of the heart muscle which lies in the left ventricle.

[35] *Anemia:* Anemia is a condition of decrease of red blood cells or hemoglobin in the blood generally resulting in limitation of the oxygen carrying capacity of the blood itself, either primary, that is, of unknown cause, or secondary to some condition such as hemorrhage or long-standing infection.

[35a] See *supra* note 30.

[36] *Myxedema:* Myxedema is a disease with a very low metabolic rate secondary to insufficiency of action of the thyroid gland.

[37] *Avitaminosis:* Avitaminosis is the result of lack of adequate vitamins in the diet or absorption thereof. It generally refers to insufficiency of vitamin B_1 such as causes beriberi.

[38] *Drugs or poisons:* Certain drugs or poisons may affect the heart by depressing its muscle through chemical effect. Thus, digitalis in great excess may poison the heart while in small doses it may act favorably to get rid of heart muscle failure. A drug called desoxycorticosterone acetate used very effectively in the treatment of Addison's disease, which is a serious disease of the adrenal glands, may in too large an amount poison the heart and cause it to dilate and fail.

Acute rheumatic heart disease occurs ordinarily in children or young adults as a result of rheumatic fever, the exact cause of which is still unknown. Usually when acute rheumatic heart disease is clearly obvious it leads to chronic heart disease, but in a minority of cases (about 20 per cent) it subsides without sequelae. *Rheumatic heart disease of the chronic type* consists of permanent structural changes in the heart and valves (including mitral stenosis)[39] caused by the inflammatory process involved in rheumatic fever. Though the cause of rheumatic fever is unknown, we do know that the rheumatic state may be excited by the hemolytic streptococcus germ[40] in susceptible persons. Thus, an epidemic of such streptococcus infection in young persons almost invariably yields a number of cases of acute rheumatism. There is a great variation in *the prognosis (probable outcome) of rheumatic heart disease* from that which is so severe that it kills in early childhood to that which causes no symptoms and but few signs,[41] and allows survival to old age, even past 80 years. The prognosis varies in general with the size of the heart, the extent of the valve lesions, and the recurrence or absence of activity of the rheumatic process.

1.b. *Relation of trauma and strain to rheumatic heart disease.* Trauma and strain do not cause rheumatic heart disease *per se* but they may perhaps predispose a person towards infection with the hemolytic streptococcus which may be followed by rheumatic fever; much more important than such factors, however, is simple contact with a carrier of the hemolytic streptococcus whereby a sore throat or other infection due to the streptococcus results. This statement is true both for the original infection and for recurrences though it has been thought that recurrences may be brought on by miscellaneous factors such as trauma; such instances, however, are rare.

[39] *Mitral stenosis:* Narrowing of the left auriculoventricular orifice through which freshly oxygenated blood returning from the lungs to the left auricle passes into the left ventricle (pumping chamber) immediately below. It is caused by inflammatory thickening and deformity of the mitral valve produced by disease, usually chronic rheumatic heart disease. The valve cusps first become thick and adherent and finally may fuse into one fibrous mass leaving only a narrow funnel-shaped, buttonhole, or fish-mouthed opening in the valve.

[40] *Streptococcus:* A genus of micro-organisms of the family *Coccaceae* including those spherical bacteria whose cells occur in chains. They are found associated with a great variety of disease conditions, among which are erysipelas, focal infections, ulcerative endocarditis, puerperal fever, septic sore throat, certain forms of enteritis, rheumatic fever, and bronchopneumonia. The streptococci are now divided into two classes: the *hemolytic,* which produce a clear zone of hemolysis (liquefaction) around the colonies on blood-agar plates; and the green-producing streptococcus (Streptococcus viridans) which produce a greenish zone about the colony on blood-agar plates. The hemolytic streptococcus is one of the most virulent germs causing infections in man.

[41] *Symptoms and signs:* These are the data which guide medical diagnosis; *symptoms* are *subjective* feelings, such as pain, which the doctor discovers by interrogating the patient; *signs,* on the other hand, are *objective* evidences of disease discoverable by medical examination without aid of the patient, as for instance, a rapid pulse, a swollen joint, or a fractured bone.

Given rheumatic heart disease acute or chronic, trauma or other strain is not likely to cause additional trouble unless the strain is severe and the heart disease considerable. Under such conditions, auricular fibrillation (rapid and incoordinate contraction of the auricles), congestive heart failure, and perhaps pulmonary and peripheral embolism[42] may ensue. But the commonest complication of all as in other kinds of heart disease, too, is a cardiac neurosis. There is an unwarranted dread of heart disease and a fear of sudden death. People must be told more about the strength and toughness of the heart and its recuperative ability.

2.a. *Hypertensive heart disease.* Hypertensive heart disease, apparently common the world over, is due to cardiac enlargement secondary to long standing or very pronounced hypertension.[43] That the high blood pressure existed long before any recent injury can usually be established by careful medical examination, particularly by finding characteristic changes in the small arteries of the eyes (abnormal eye grounds).[44] Congestive heart failure is a common late complication, beginning with engorgement of the lungs and dyspnea (labored breathing), acute or chronic, when the left heart fails,[45] and going on to engorgement of liver and of neck veins and dependent edema (swelling in dependent parts), in the legs for instance, when the right heart fails secondarily to failure of the left.[46] *The prognosis (probable outcome) of hypertensive heart disease* varies greatly but in general is unfavorable after the appearance of clearcut signs of cardiac enlargement or abnormal electrocardiogram, a few years intervening after that before death occurs

[42] *Embolism:* The plugging of an artery or vein by a clot or obstruction which has been brought to its place of lodgment by the blood current. Pulmonary embolism refers to such plugging of a main artery in the lung; peripheral embolism involves plugging of an artery in one of the extremities by the same mechanism.

[43] *Hypertension:* High blood pressure causes cardiac enlargement in the course of time chiefly by the increased work imposed on the heart muscle. The left ventricle is more or less under constant strain, tends gradually to dilate, and the eventual reaction to the dilatation is an increase in muscle mass called hypertrophy.

[44] *Abnormal eye grounds:* Abnormality of the eye grounds (see note 18, *supra*) is common in hypertension. The small arteries of the retinae tend, after years of high blood pressure, to become narrow and tortuous and these conditions can be readily seen by the ophthalmoscope. By far the commonest cause of such changes in the arteries of the eyegrounds is high blood pressure.

[45] *Failure of left heart:* Failure of the muscle of the left ventricle finally results after long continued or very excessive strain on the left ventricle, especially in cases of high blood pressure. Enlargement of the left ventricle always precedes its failure, whether the enlargement consists of simple dilatation or preponderant hypertrophy or both dilatation and hypertrophy.

[46] *Failure of right heart:* After the left ventricle dilates it fails to pump out all the blood it receives and the blood vessels of the lungs are engorged as a result. Thus there ensues an increase in blood pressure in the pulmonary circulation. This increase in blood pressure in the pulmonary circulation causes an increase in the work of the right ventricle and eventually under that strain the right ventricle also tends to enlarge and to fail.

from congestive heart failure, coronary complications,[47] apoplexy,[48] or uremia,[49] unless the strain is reduced, as may sometimes be accomplished by an invalid life or in selected cases by surgically cutting certain of the sympathetic nerves in the back (so-called *lumbodorsal splanchnic resection*).[50]

2.b. *Relation of trauma and strain to hypertensive heart disease.* From the foregoing the reader will see that neither injury nor strain can be incriminated scientifically as the cause of high blood pressure and consequent hypertensive heart disease.[51] The cause of hypertension (high blood pressure) is usually unknown; medical men often refer to such cases as "essential hypertension." If the individual has only slight hypertension or slight hypertensive heart disease, slight to moderate trauma or strain does no harm. If, however, his condition is far advanced and the heart is already considerably enlarged, congestive heart (left ventricular) failure with pulmonary congestion and edema may be precipitated by severe strain.

3.a. *Coronary heart disease.* Coronary heart disease, also quite common the world over, is seen most frequently in middle aged men under very little physical strain; it seems to be relatively rare in farmers and laborers. The disease is due to *atherosclerosis* of the coronary arteries,

[47] *Coronary complications:* An important complication of hypertensive heart disease results from arteriosclerosis of the coronary arteries which is commonly found in such cases; in fact arteriosclerosis generally is hastened by the presence of high blood pressure. If the coronary arteries, either independently or secondarily, become too narrow in hypertensive heart disease, angina pectoris may result, or even actual coronary thrombosis, that is, clotting of blood in one of the coronary arteries resulting in infarction of the myocardium.

[48] *Apoplexy:* Apoplexy, that is, hemorrhage into the brain with paralysis or death resulting, is most commonly found in persons who have had high blood pressure for years.

[49] *Uremia:* Uremia is a poisoning of the system secondary to insufficiency of kidney action, most commonly found either as a result of infection of the kidneys, that is, nephritis, or secondary to chronic high blood pressure.

[50] *Splanchnic resection:* Cutting of the splanchnic nerves has been introduced as a treatment of high blood pressure because it tends to abolish constriction of blood vessels below the diaphragm, including those to the kidneys, such constriction being at least partly responsible for the hypertension; an important secondary factor may be an improvement in the circulation of the kidneys themselves.

[51] *See* Malloy v. Southern Cities Distributing Co. (La. App.) 142 So. 718 (1932). Due to defendant's negligent maintenance of its gas main, an explosion occurred in the pharmacy of which plaintiff was a managing partner. He was thrown from the mezzanine to the main floor seven feet below. In an action for damages, plaintiff contended that his heart had been seriously affected, in that it now raced, and that the explosion caused high blood pressure. P's attending physician, X, erroneously supported these contentions by expert testimony at the trial; he attributed P's symptoms to degeneration of the heart muscle caused by the explosion (an invalid hypothesis) and the jury returned a verdict for P for $4,000. Judgment was affirmed on appeal. Such a stimulus could not cause high blood pressure; that P had long been subject to it is shown by X's testimony that he found ". . . some enlargement of the heart." That ". . . it was not unusual for P's heart rate to change without warning in 5 or 6 seconds from 72 to 122" shows a mild degree of *paroxysmal tachycardia* (fast heart rate, discussed in text *infra*), not serious in absence of congestive failure.

the vessels which supply the heart with blood. *Atherosclerosis* is a degenerative process[52] which tends to narrow the lumen (inner diameter) of the coronary artery involved, either by fibrotic changes, by cholesterol sacs, or by calcareous plaques, and so to reduce the blood supply to the heart (see Fig. 3). The patient's history is likely to show the presence of *angina pectoris.*[53] There is always a risk of *acute myocardial infarction* (localized heart muscle destruction with replacement by scar), due to coronary thrombosis (obstruction of an important coronary artery by a blood clot (thrombus); such an occurrence is always serious and infrequently it is quickly fatal. *The prognosis of coronary heart disease* must always be guarded, sudden death being possible at any time from further trouble (angina pectoris or acute coronary thrombosis). However, most patients live for years and may, with the development of an adequate collateral coronary circulation,[54] reasonable care to avoid unnecessary physical or nervous strain, and good fortune, recover completely from their symptoms and signs and even be perfectly well 10, 15 or 20 years later. In fact, a careful long time follow-up study of a large group of cases of angina pectoris (approximately 500), has led us to raise the average life expectancy following the first symptom from an old estimate of 4½ to 5 years to a new one of 9 to 10 years.[55] Similarly, acute coronary thrombosis no longer has so dismal a prognosis as it once had now that we have followed the course of many victims over a more adequate period of time.

3.b. *Relation of trauma and strain to coronary heart disease.* There is no indication that physical trauma or strain can cause coronary disease, which as we said is due to *atherosclerosis;* whether long continued mental stress may favor the onset, is not quite so clear, but there is no

[52] *Atherosclerosis:* The cause of this degenerative process is unknown: nervous strain, the tension of modern city life, infection, wear and tear, overnutrition and a high cholesterol diet have been blamed in turn but the fact is that we do not know what causes early or excessive coronary arteriosclerosis; one of the least likely factors is trauma.

[53] *Angina pectoris:* Angina pectoris is a symptom of insufficiency of the circulation of the blood to the heart muscle through the coronary arteries. It consists of a sensation of oppression under the breastbone, coming particularly on effort, lasting a few minutes, and relieved by rest or nitroglycerine. The pain may or may not radiate down the arms.

[54] *Collateral coronary circulation:* The collateral coronary circulation consists of anastomotic vessels, few or small in the case of normal hearts, but much increased in size in the presence of slowly developing coronary heart disease, whereby blood can be rerouted through undamaged vessels to parts of the heart muscle not adequately supplied through their original coronary vessels because of disease therein. It is often possible for collateral circulation to develop adequately ahead of sclerosis and narrowing of the vessels so that no harm to the heart muscle results at all, or it may happen that the collateral circulation develops only after damage has already resulted.

[55] WHITE, P. D.; BLAND, E. F. AND MISKALL, E. W., *A Long Time Follow-Up of the Prognosis of Angina Pectoris (497 Cases), Including a Note on Angina Pectoris Decubitus.* 123 JOUR. A. M. A. 801 (1943).

proof thereof. Experience shows that the majority of individuals with *angina pectoris* (which is a symptom of coronary disease), and with actual coronary thrombosis, have been mental workers, but the probability of a causal connection is not established medically since it is difficult to assess accurately the etiological role of other factors such as the eating of rich foods without adequate physical exercise, disorders of fat metabolism, familial and racial tendencies to early arteriosclerosis, and the like. We would be safe in saying that there is no medical evidence that isolated emotional or nervous stimuli of a transient sort such as might be caused by sudden fright or other medicolegal stimuli, could precipitate the onset of coronary disease, though such might be brought to light thereby.

Assume, however, that the individual had pre-existing coronary disease of an advanced sort: could physical or psychic trauma precipitate a cardiac disaster? The exact way in which thrombotic occlusion of an atherosclerotic coronary artery takes place is not known. Given coronary heart disease, however—especially with coronary insufficiency—trauma or strain, physical or nervous, may aggravate the condition and on very rare occasions may even be followed by acute coronary thrombosis[56] and myocardial infarction[57] (see Fig. 4). As the great majority of acute coronary occlusions in persons with coronary disease occur at rest, without exertion or trauma, the alleged causal connection should be subjected to close scrutiny. Did the traumatic episode actually precede the appearance of the cardiac disaster? Or did the claimant have a heart attack during the course of work, not as a result of accidental injury, and then fall and suffer injury? These events must be reconstructed with careful efforts to determine the correct chronology and the nature and intensity of the traumatic stimulus.

Sudden death is, on the other hand, readily and commonly caused by severe physical strain in the presence of coronary insufficiency with angina pectoris easily induced (because the work placed on the heart is greater than its impaired blood supply can support); nervous or emotional strain is much less likely to do this but may also be responsible.[58] Angina pectoris, due to trauma or strain, usually appears dur-

[56] *Acute coronary thrombosis:* Formation of a clot in a branch of the coronary arteries which supply blood to the heart muscle, resulting in sudden obstruction of the artery.

[57] *Myocardial infarction:* Death of a localized area of heart muscle due to loss of its blood supply from obstruction of circulation to the area as by coronary thrombosis.

[58] Court cases are numerous involving death allegedly caused by severe physical strain in the presence of coronary insufficiency. For cases involving alleged injury to the heart precipitated by nervous or emotional strain (psychic stimuli) see the following: Currie v. Wardrop, (Scotland, 1927) S. C. 538, (1927) Scot. L. T. 383 (Tachycardia and tremors); Kelly v. Lowney & Williams, 126 Pa. (2d) 486 (Mont. 1942) (Heart failure); Rasmussen v. Benson, 133 Neb. 449,

ing the strain or immediately thereafter. A few minutes, a few hours, or a day or two may, however, elapse after some fall, or blow, or other physical strain before typical coronary occlusion may set in. A long time interval between the trauma and the appearance of coronary thrombosis should rule out a causal connection; arbitrarily, one may set this interval at several days, certainly not more than one week; however, the interval may perhaps be extended to two weeks in cases in which coronary thrombosis develops following a severe injury that confines the patient to bed for many days. Even in such cases, coronary thrombosis that appears more than two weeks after the injury should be regarded medically as a coincidence. The Workmen's Compensation laws of many states authorize the Commission to order a postmortem examination where the cause of a workman's death is obscure; in all cases of suspected death from coronary thrombosis, autopsy is desirable to provide evidence for or against the hypothesis.

We shall see subsequently, in discussing pervasive medicolegal aspects of heart conditions, that a heart sufficiently impaired by any cause or disease, may prove unable to withstand particular trauma; here the trauma may precipitate myocardial (heart muscle) insufficiency, even to the degree of extreme congestive failure, and thus lead to serious injury or death by a mechanism distinct from coronary occlusion or thrombosis. (See discussion *infra.*)

A final question concerning the relationship of coronary disease to trauma is this: May coronary occlusion due to embolism (plugging of the coronary artery by a clot or obstruction carried there by the blood stream) follow trauma? The answer is: "Probably yes, but only in the rarest cases." Coronary embolism is excessively rare: when it does occur it is much more often due to disease than to injury.[59] It follows that any hypothesis of coronary embolism due to trauma should be viewed as medically improbable, unless there is very strong positive evidence, preferably based upon an adequate postmortem examination.

Assume that the plaintiff or claimant has suffered an acute myo-

275 N. W. 674 (1937), aff'd., 280 N. W. 890 (1938) (Heart failure); Newton v. New York N. H. & H. R. Co. *et al.*, 106 App. Div. 415, 94 N. Y. Supp. 825 (1905) (Heart failure); Hoage v. Royal Indemnity Co. (Ct. of App., D. C.), 90 F. (2d) 387 (1937) (Workmen's Comp. case: angina pectoris and coronary thrombosis imputed to excessive nervous strain from long hours of clerical work, without traumatic injury). Some of the cases involve both physical exertion and psychic stimuli, as in Rasmussen v. Benson, *supra* and in the following workmen's compensation cases: Boest v. 1318 Kings Highway Corporation, 268 App. Div. 943, 51 N. Y. S. (2d) 353 (1944), and Godsman v. Grumman Aircraft Engineering Corp., 268 App. Div. 945, 51 N. Y. S. (2d) 368 (1944).

[59] *Coronary embolism:* When coronary embolism does occur, it is usually the result of bacterial endocarditis involving the aortic or mitral valve; or of the spontaneous breaking off of a bit of blood clot (thrombus) formed previously in the left auricle or the left ventricle. The possibility of traumatic embolism is a much more remote one according to medical experience.

cardial infarction (death of a portion of the heart muscle due to sudden loss of its blood supply) as a result of adequate trauma acting upon his advanced coronary disease. He will naturally go to bed. Electrocardiograms should be made at intervals as these give a running picture of the nature and degree of this species of heart trouble. During the first two weeks of acute myocardial infarction before the scar is well set, rupture of the patient's heart may occur with serious or fatal results in event of too much activity and excitement.[60] This may happen more readily in the case of insane patients or of cases inadequately treated; quinidine sulphate[61] in daily rations and absolute bed rest help to prevent such cardiac disasters.

4.a. *Congenital heart disease.* Congenital heart disease involves a cardiac abnormality existing from birth. It is a rare type of heart disease fortunately, not accounting for more than 2 or 3 per cent of cardiac patients. The diagnosis may be difficult to establish because of the many complicated combinations of developmental defects which may occur, but frequently the abnormality will have been detected soon after birth or within the first year or two of life. Cyanosis (bluish coloration of the skin), and clubbing of the fingers[62] noted since early childhood, with an abnormal heart and without pulmonary (lung) disease sufficient to cause such changes, is fairly good proof. *The prognosis of congenital heart disease* varies enormously from death at birth in case of extreme cardiovascular anomalies to good health in old age in cases of the lesser defects such as coarctation of the aorta[63] and patency of the ductus

[60] *Rupture of heart wall:* The rupture of the heart wall allows blood to escape into the surrounding pericardial sac (hemopericardium), the pressure thus built up "smothers" the heart by impeding its normal contractions (cardiac tamponade), and this may cause rapid death. Another mechanism which may lead to rapid death in the case of myocardial infarction is ventricular tachycardia due to gross irritability of the ventricular muscle; such a disorder of rhythm may change to irregular vermiform beating of the ventricles (ventricular fibrillation) and this ordinarily results in speedy death.

[61] *Quinidine sulphate:* Quinidine is an alkaloid derived from cinchona bark. It is isomeric with, and its taste, appearance, uses, and dose are the same as those of quinine, although its effect on the cardiac muscle is more pronounced, depressing excitability and conduction in the auricular muscle. It is used in the form of the sulphate or bisulphate in the treatment of auricular fibrillation and other cardiac arrhythmias.

[62] *Clubbing of the fingers:* Clubbing of the fingers consists of the bulbous enlargement of the ends of the fingers found in a few disease conditions and rarely as a familial anomaly without disease. The conditions in which clubbing of the fingers are most commonly found are: (1) Chronic serious disease of the lungs; (2) certain congenital heart abnormalities with cyanosis; and (3) subacute bacterial endocarditis, that is, inflammation of the heart lining and valves usually caused by the Streptococcus viridans.

Since pulmonary disease is the commonest cause of clubbing of the fingers, this must be ruled out before we can accept the finding as indicative of heart disease.

[63] *Coarctation of the aorta:* Coarctation of the aorta consists of a congenital narrowing of the aorta usually in the neighborhood of the insertion of the ductus arteriosus; this narrowing of the aorta increases the blood pressure in the upper

arteriosus.[64] In general, the cyanotic cases (with the morbus caeruleus *(congenital cyanosis—i.e., bluish coloration of skin) or maladie bleue)* die young, rarely surviving the age of 25 years; there are a few notable exceptions reaching well into middle age who show the most common type of the *morbus caeruleus,* namely the tetralogy of Fallot.[65]

4.b. *Relation of trauma and strain to congenital heart disease.* Congenital heart disease is, of course, in no way related to trauma or strain except as such factors may precipitate rare complications—heart failure, cerebral thrombosis,[66] or sudden death.

5.a. *Cardiovascular syphilis.* Syphilis is an infectious disease caused by an organism called the *treponema pallidum.* It is most often acquired, rarely congenital, and may affect various tissues of the body, including the heart and blood vessels. The *acquired form* of syphilis is practically always due to sexual exposure and involves first an initial sore and an early stage of blood stream invasion with relatively mild disturbance of the patient's system, with eruptions appearing upon the skin and mucous membranes. After a variable time interval of months to years, there follows a late stage in which the disease causes connective tissue elements to increase with coincidental destruction of some other tissue elements and the patients may then note a consequent disturbance in the functions of various organs. The *congenital form* of syphilis has similar ultimate effects but does not involve any primary lesion and the late manifestations predominate. It has been estimated that 10 per cent of adult Americans at some time are infected with syphilis but the incidence varies enormously among the racial and social groups, often running as high as 25 per cent among poor Negroes and less than one per cent in groups of high culture. In studying 1,000 syphilitic patients, Cochems and Kemp found cardiovascular syphilis in 10.8 per cent of the women and 13.4 per cent of the men, with a gross incidence of 12.7

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part of the body with a decrease of blood pressure in the lower part. This increase in blood pressure acts as a strain on the left ventricle which is usually enlarged; a condition similar to that of hypertensive heart disease tends to result.

[64] *Patent ductus arteriosus:* The ductus arteriosus is an arterial trunk which in the fetus carries blood directly from the pulmonary artery into the aorta and systemic circulation without the blood first passing through the lungs. There is no need for the blood to go to the lungs for oxygenation before birth, for the fetus derives its oxygenated blood from the mother and its own lungs are virtually nonfunctional. Normally this fairly large arterial trunk closes at, or shortly after, birth but it may remain open thus producing an important congenital defect of the cardiovascular system.

[65] *Tetralogy of Fallot:* A grouping of congenital cardiac defects commonly found in adults, namely: pulmonary stenosis; interventricular septal defects, dextroposition of the aorta (so that it receives blood from the right as well as from the left ventricle) and a large right ventricle (hypertrophied from overwork). This grouping of congenital cardiac defects was described by Fallot in 1888.

[66] *Cerebral thrombosis:* A plug or clot of blood formed in one of the vessels which supply the brain.

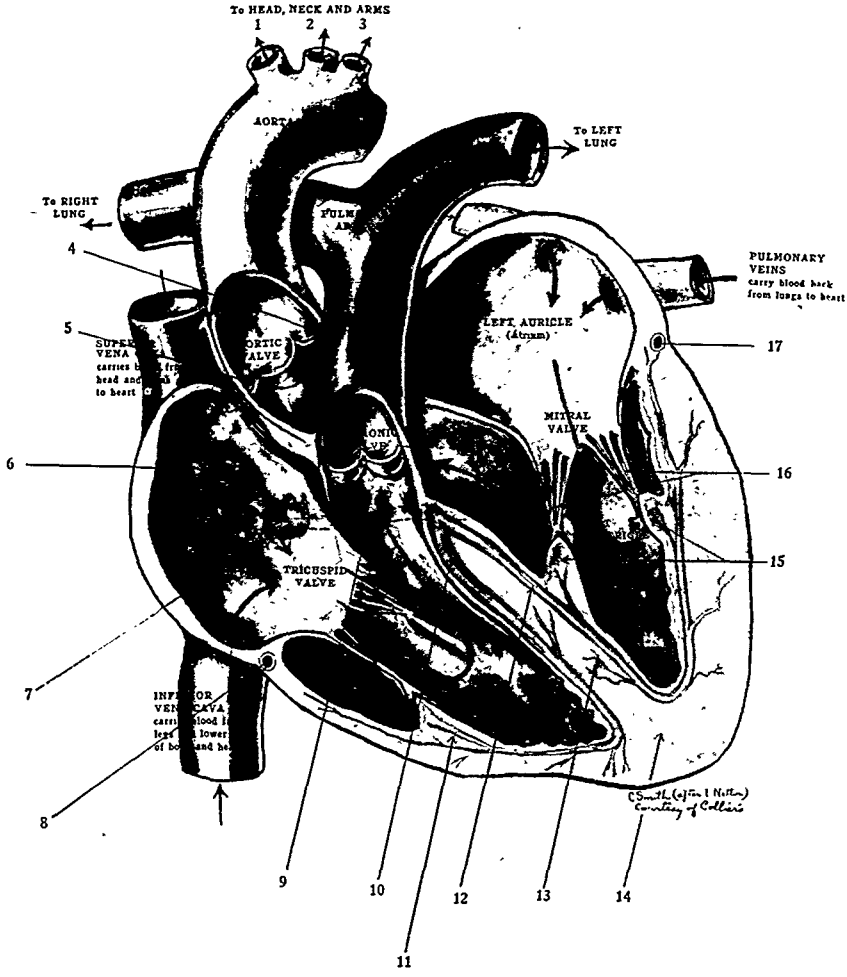

FIGURE 1. DRAWING OF A DISSECTION INTENDED TO SHOW MAIN ANATOMICAL AND PHYSIOLOGICAL FEATURES OF THE HEART AND THE GREAT VESSELS

1 = Innominate Artery
2 = Left Carotid Artery
3 = Left Subclavian Artery
4 = Left Coronary Artery
5 = Right Coronary Artery
6 = Sino-auricular node ("Pace-maker" of the heart)
7 = A.-V. Node (of Tawara)
8 = Cross section of right coronary artery
9 = A.-V. Bundle (of His)
10 = Right bundle branch
11 = Purkinje network
12 = Left bundle branch
13 = Interventricular septum
14 = Apex of heart
15 = Papillary muscles
16 = Chordae tendineae
17 = Cross section of circumflex branch of left coronary artery

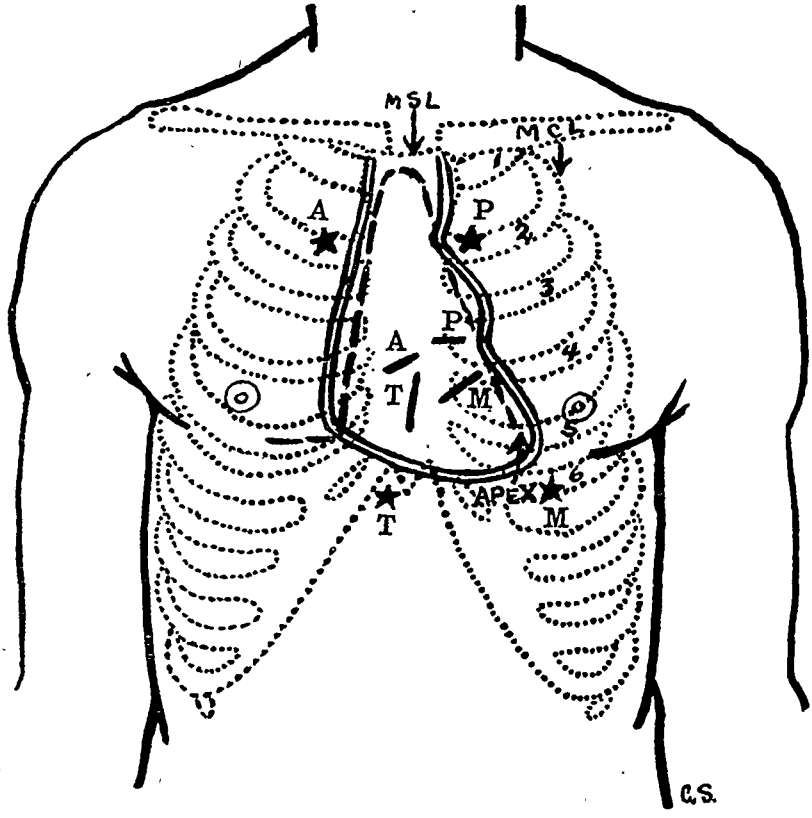

FIGURE 2

DIAGRAM OF ANTERIOR CHEST WALL SHOWING RELATIVE POSITIONS OF HEART BORDERS BY PERCUSSION (BROKEN LINE) AND BY ORTHODIAGRAM (DOUBLE LINE)

Dotted lines outline postions of ribs

Estimated positions of valves:

A = Aortic
P = Pulmonary
T = Triscuspid
M = Mitral

★ = Points where the individual valve sounds and murmurs are best heard
★A = Point where aortic valve sounds are best heard
★P = Point where pulmonary valve sounds are best heard
★T = Point where triscuspid valve sounds are best heard
★M = Point where mitral valve sounds are best heard

MSL = Mid sternal line
MCL = Mid clavicular line

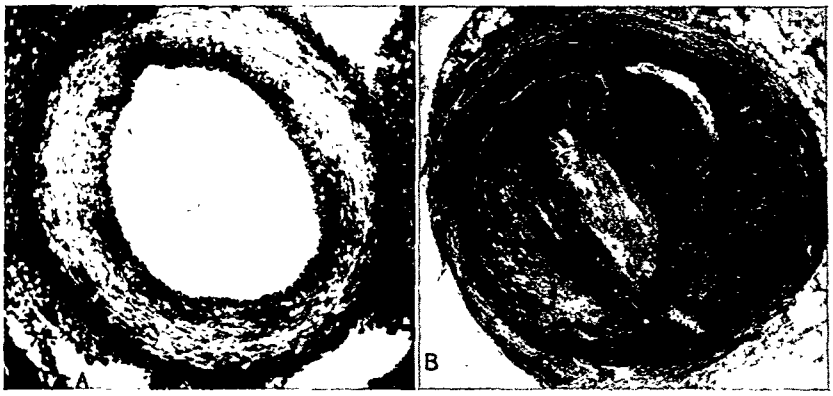

FIGURE 3

A. Microphotograph of normal coronary artery. (Kindness of Dr. Benjamin Castleman.)

B. Microphotograph of coronary artery in youthful case of extensive coronary atherosclerosis. Man aged 41 years collapsed while walking to work and died a few minutes later. Note the extensive fibrosis of the intima obliterating the lumen except for a small crescentic space near the media at the right upper border. (Kindness of Dr. Timothy Leary.)

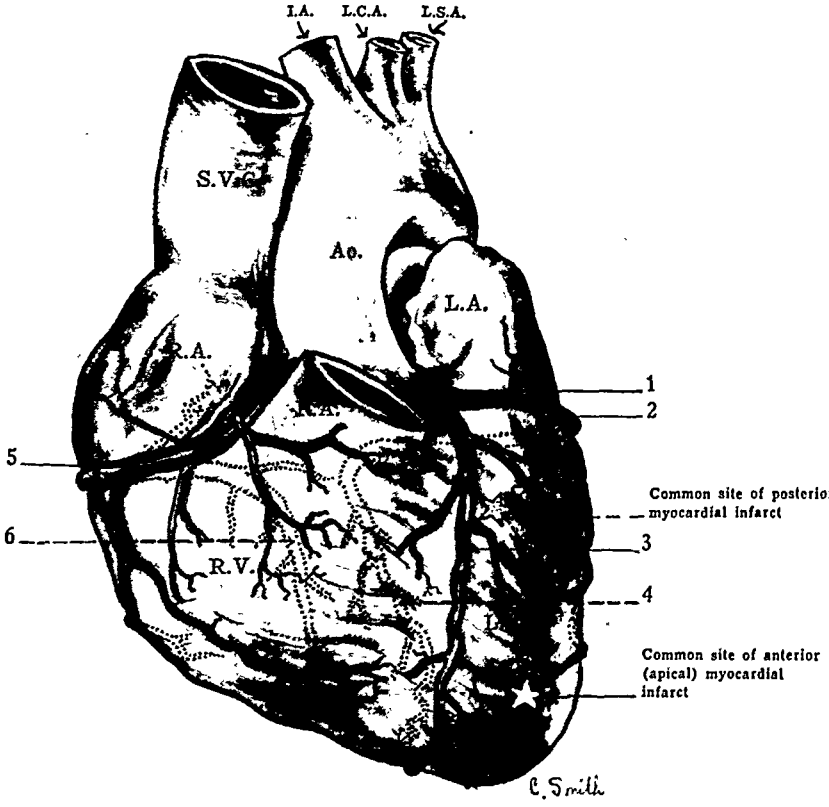

FIGURE 4. DIAGRAM OF CORONARY ARTERIES

1 = Main trunk of left coronary artery
2 = Circumflex branch of left coronary artery
3 = Anterior descending branch of left coronary artery
4 = Posterior descending branch of left coronary artery
5 = Right coronary artery
6 = Posterior descending branch of right coronary artery

S.V.C. = Superior Vena Cava
Ao. = Aorta
R.A. = Right Auricle
L.A. = Left Auricle
R.V. = Right Ventricle
L.V. = Left Ventricle

NOTE: Broken and dotted lines indicate posterior surface.

per cent. At present cardiovascular syphilis is rapidly decreasing in the more civilized parts of the world.

Cardiovascular syphilis becomes evident, as a rule, in the forties or fifties. In general, the main injurious effects are the following:

1. *Inflammatory changes (syphilitic aortitis) most often occur in the aorta,* the large artery which is the main conduit conveying freshly oxygenated blood from the left ventricle to the body tissues. The *aortic valve* separates the left ventricle from the aorta and just beyond, away from the heart, are the openings (mouths) of the coronary arteries which convey blood from the aorta to the heart muscle. Now the inflammatory changes in the aorta which occur in *syphilitic aortitis* usually begin in the aorta just above the valve and later extend down to the mouths of the coronary arteries and to the valve cusps (leaves) with the following possible results:

(a) *Aortic regurgitation* may develop from inability of the aortic valve to close completely the orifice between the left ventricle and the aorta. This may be due to degenerative changes in the valve or to stretching of the aorta at the point where the valve is attached to it (weakening and dilatation of the aortic ring), or to both. The regurgitation of blood decreases the efficiency of the heart as a pump and increases its work.

(b) Degenerative changes due to the inflammatory reaction may so weaken the walls of the aorta that pressure causes a localized ballooning of the artery wall: this is called an *aneurysm* and it obviously involves a progressive risk of rupture.

(c) The inflammatory reaction in the aortic walls may cause *partial or almost complete blockage of the mouths of the coronary arteries* so that the hard working heart muscle is unable to get its essential supply of freshly oxygenated blood.

2. *Inflammatory changes due to direct involvement by syphilis may rarely appear in the myocardium* (heart muscle) and this may entail progressive fibrous replacement of the muscle fibers. The same symptoms occur as follow myocardial (heart muscle) disease from other causes, except that cardiac failure may come with considerable abruptness and that disturbances of rhythm are relatively rare.

Thus, the commonest complications of aortic syphilis are congestive heart failure secondary to aortic regurgitation, rupture of an aneurysm with sudden death, and coronary insufficiency due to blocking of the coronary artery mouths by the inflammatory reaction in the aortic wall. The *prognosis in cardiovascular syphilis* is especially bad and must at

the outset be guarded; the less the physical strain and the more adequate the early or even later anti-syphilitic treatment,[67] the longer the patient may be expected to live.

5.b. *Relation of trauma and strain to cardiovascular syphilis.* The reader will appreciate the fact that injury or strain can have no primary causal connection with the degenerative changes which in syphilis result from the inflammatory process set up by the *treponema pallidum;* the medicolegal interest, therefore, is focused upon whether and how injury or strain may act upon the established condition to aggravate it or to accelerate death. We have seen that the disease may cause weakness of the aortic wall with aneurysmal dilatation and that the strain on the heart is increased by the presence of *aortic regurgitation* or *coronary mouth occlusion* or by both. These all operate to make the cardiovascular system very subject and sensitive to strain, especially physical, which may cause one of the following results, namely:

(1) Sudden death by rupture of an aortic aneursym;

(2) Sudden death from overwhelming coronary insufficiency;

(3) Acute dilatation of the left ventricle, with extreme congestion of the lungs with blood (pulmonary edema) from which the patient may die or recover.

One must remember that all these complications of cardiovascular syphilis are common with no obvious strain at all. Therefore, in establishing a traumatic causation, it is necessary to prove an adequate exertion or strain followed within a reasonable time by symptoms of injury or cardiovascular disaster, and in case of death, the evidentiary aid to be had from a postmortem examination is of much value.[68]

6. *Bacterial endocarditis.* Bacterial endocarditis (bacterial inflammation of the *endocardium,* the lining membrane of the heart) may be *acute* (i.e., runs a short and relatively severe course), or *subacute* (i.e., runs a longer course, of 2 months or more, averaging 6 months). The acute variety as a rule is merely a complication[69] of some other primary

[67] *Antisyphilitic treatment:* Antisyphilitic treatment consists of the use of certain chemicals, in particular bismuth, arsenic, and mercury, which control and destroy the treponema pallidum, the organism which causes the syphilitic infection. Much of this treatment is given by intramuscular or intravenous injection. At present penicillin is a favored drug for treating the disease in its acute stage but not in its later cardiovascular manifestations.

[68] Thus, in Keely v. Metropolitan Edison Co., 177 Pa. Super. 63, 41 A. (2d) 420 (1944), X's chest was bruised by the steering wheel when the car he was driving in course of his employment overturned. He complained of chest pain but worked for a week before he suddenly collapsed and died. A post-mortem examination proved conclusively that the cause of death was an injury received in the automobile accident. The laws of many states give workmen's compensation commissioners discretionary power to order post-mortem examinations when the cause of death is obscure.

[69] *Complication:* The concurrence of two or more diseases in the same patient.

septic[70] or pneumonic (lung) infection and it may be unrecognized or unrecognizable during life. In this age of sulfonamide and penicillin treatment it has become far less common. Subacute bacterial endocarditis is fortunately not common; it is due to the grafting of an acute Streptococcus viridans[71] infection on thrombi[72] laid down on heart valves roughened and scarred by chronic rheumatic heart disease and on congenital defects in the heart or great arteries.[73] The bacteria reach the heart through the blood stream but how they get into the blood is usually not known. It is likely that all of us harbor in our circulations such relatively innocent invaders off and on for short periods of time without harm. The onset of the disease is insidious and hard to date. The disease is a long and tiresome one, in the past usually fatal, even with sulfonamide therapy, but now curable in the majority of cases (about 75 per cent) by the use of penicillin in adequate dosage. In fatal cases the disease lasts on the average six months before death ensues. Subacute bacterial endocarditis is recognizable medically by its clinical course (persistent daily fever for weeks to months without other explanation), evidence of chronic heart disease, enlarged spleen,[74] clubbing of the fingers,[75] petechial hemorrhages,[76] and positive blood cultures.[77] Rarely are all these findings present in the same case but any three are sufficient evidence to support the diagnosis.

6.b. *Relation of trauma and strain to bacterial endocarditis.* Acute bacterial endocarditis, as we have seen, may occur as an incident of any

[70] *Primary sepsis:* The original focus or point of origin of a putrefactive infection. Septic refers to an acute virulent infectious process anywhere in the body resulting generally from potent germs such as the streptococcus, staphylococcus, or pneumococcus.

[71] *Streptococcus viridans:* That main class of the coccus which produces a greenish zone about the colony on blood-agar plates; it is not so virulent as the hemolytic streptococcus.

[72] *Thrombus:* A blood clot which remains where it is formed in a blood vessel; or in the heart; if it moves on in the blood stream to a new site it becomes an *embolus.*

[73] Bacterial vegetations form which are composed entirely of fibrin and bacteria; they become firmly attached to the heart valves or other sites.

[74] *Enlarged spleen:* Enlargement of the spleen is an important indication of disease; there are a number of different diseases in which it may appear, more commonly malaria, typhoid fever, cirrhosis of the liver, and certain blood diseases; less commonly, the condition herein described, namely, subacute bacterial endocarditis.

[75] *Clubbing of the fingers:* Bulbous enlargement of the ends of the fingers found in a few disease conditions and rarely as a familial anomaly without disease. It is most commonly associated with chronic serious disease of the lungs, with certain congenital heart anomalies with cyanosis (bluish coloration of the skin) or with subacute bacterial endocarditis.

[76] *Petechial hemorrhages:* Petechial hemorrhages are minute areas of bleeding anywhere in the body seen most commonly under or in the skin or conjunctivae or mucous membranes.

[77] *Positive blood cultures:* Bacteriological cultures from the blood are considered positive when some bacterium is constantly grown from blood samples smeared on a proper culture medium. The organisms most commonly found are the streptococcus, the staphylococcus, the pneumococcus and the meningococcus.

overwhelming bacterial infection whether due to trauma or not. Its medicolegal implications will depend, therefore, on whether the primary bacterial infection arose from some culpable or compensable cause[78] and probably (based on course of symptoms, time relations, etc.) gave rise to the acute endocarditis as a sequel.[79] Quite often it will not be necessary for the lawyer or court to be concerned with the causal connection for the reason that the primary infection (sepsis) was so overwhelming as to cause death independently of the endocarditis. It is difficult to diagnose acute bacterial endocarditis, and it is usually discovered as a postmortem finding. Acute bacterial endocarditis is most commonly caused by the following bacteria: the Streptococcus hemolyticus, the Staphylococcus aureus, the Staphylococcus albus, the pneumococcus, and the gonococcus; in very rare cases it is caused by a few other organisms.

In considering the medicolegal aspects of *subacute bacterial endocarditis,* one must recall that the disease usually begins insidiously without any history of recent trauma or even of focal infection, in a heart already damaged, usually by rheumatic lesions and less often by congenital defects. In perhaps one-quarter to one-third of the cases tooth extractions seems to be responsible and very rarely some accident or strain has preceded the beginning of signs and symptoms of the disease as in the case to be related shortly. Oftentimes, careful medical study will show that the endocarditis antedated the trauma and that no reason exists for assuming that any injury aggravated or accelerated the condition. In a very few cases the medical man may be warranted in assuming that trauma, with or without infection, has predisposed to the subacute bacterial endocarditis, perhaps by lowering the resistance of the patient. As a rule, however, an unsupported medical inference should not be accepted that the responsible Streptococcus viridans actually entered the body through the wound for medical experience is to the contrary; other bacteria, if any, are usually found in cultures from wounds. The Streptococcus viridans apparently is a common invader of the blood stream, even in healthy individuals, and if the heart valves are normal and the body resistance satisfactory, endocarditis does not

[78] Under *tort* law, whose principles govern personal injury actions between strangers, culpability is required, consisting of willful or negligent behavior by the defendant intended or calculated to injure another person in plaintiff's position. Workmen's compensation law, however, provides benefits without proof of culpability: it is enough to show that the workman's injury or disease, or death, was caused, aggravated or accelerated by an accident arising out of and in the course and scope of employment.

[79] *See* D'Amico v. Middlesex Dress Corporation, 132 N. J. L. 116, 38 A. (2d) 857 (1944) (Death not compensable: due to independent sub-acute bacterial endocarditis not related to fall down employer's stairs); Maynard v. B. F. Goodrich Co., 144 Ohio St. 22, 56 N. E. (2d) 195 (1944) (X died of bacterial endocarditis; held: whether injury, in course of employment, was proximate cause of death, under the evidence was for the jury.).

develop. Once the disease is in progress, aggravation by any special factor is unlikely. The following case may be of interest as indicating the type of evidentiary problems one may encounter.

Complicating Disease of an Already Damaged Heart, Coincidental with Trauma: Subacute Bacterial Endocarditis Superimposed on Chronic Rheumatic Valvular Disease.[80] Gleason cites the case of a man, aged about thirty-seven years, who had been in good health for several years previous to the injury. On July 15, 1924, he stepped on a nail injuring his left foot. The attending physician operated on the foot, but there was, apparently, no septic condition present. The patient was out of work for one week; he then resumed work for two weeks, wearing a slit shoe. After his return to work the foot gave him no trouble. He was then laid off for three weeks. At about the middle of August, he began to have a fever every afternoon. When he returned to work for the second time, about six weeks after the injury, he noticed that his right hand was swollen. He worked intermittently until the first of September, although the increased afternoon temperature persisted. He died in August, 1925, the cause of death being subacute bacterial endocarditis.

The patient, prior to his injury, had a well-compensated chronic rheumatic valvular heart disease, which had been causing him no physical disability. This heart disease was probably due to repeated attacks of rheumatic fever in early life. It was agreed that the condition which developed in August, following the injury, was subacute bacterial endocarditis superimposed upon chronic rheumatic valvular disease.

A culture of the blood,[81] made in June, 1925, revealed Streptococcus viridans, the organism usually found in subacute bacterial endocarditis, and not ordinarily found in septic infections from an external wound.

The medical testimony was divided as to whether the injury, which was not septic, brought about the subacute bacterial endocarditis which resulted in death. One of the physicians stated that a trivial injury may initiate a fatal illness in a person suffering from rheumatic heart disease.

7.a. *Thyroid heart disease.* Thyroid heart disease is rare and is growing still rarer, due to the early recognition and adequate treatment in these days of both *thyrotoxicosis*[82] and *myxedema.*[83] The thyro-

[80] GLEASON, G., *Accidents and Heart Disease from the Insurance Company's Point of View,* 3 AM. HEART J. 525 (1928).

[81] *Culture of the blood:* Blood cultures are made by drawing a few cubic centimeters of blood from an arm vein and planting them in special culture media which may be broth or agar (a type of gelatine).

[82] *Thyrotoxicosis:* Thyrotoxicosis is a toxic state of the body resulting from overaction of the thyroid gland. It is synonymous with hyperthyroidism and Graves' disease. Thyrotoxicosis is characterized by excessive nervousness, fast heart rate, prominence of the eyes (exophthalmos), increased perspiration, loss of weight, and marked elevation of the basal metabolic rate (B.M.R.).

Thyroidectomy consists of surgical removal of the thyroid gland; sub-total thyroidectomy is the operation performed for thyrotoxicosis and it involves only partial excision of the gland.

[83] *Myxedema:* Myxedema is a disease resulting from lack of thyroid secretion and is characterized by extreme sluggishness and sensitiveness to cold, dry skin, loss of hair, and very low basal metabolic rate.

toxic or hyperthyroid heart is overactive in rate and force and in time may be the victim of auricular fibrillation and eventually of enlargement and failure, unless the patient receives treatment with iodine and as a general rule submits to the partial removal of the thyroid gland by surgical operation *(sub-total thyroidectomy)*. The myxedema heart is an enlarged, sluggish heart which produces a characteristic electrocardiogram.[84] The condition is correctible by thyroid therapy[85] which must be administered carefully to avoid raising the metabolism[86] of body and of heart so high or so rapidly as to induce coronary insufficiency (inadequate blood supply) in a heart muscle often receiving insufficient blood because of disease of the coronary arteries.

7.b. *Relation of trauma and strain to thyrotoxic heart disease.* The reader will perceive that the cause of *thyrotoxic heart disease* is chronic, severe, untreated thyrotoxicosis resulting from excessive secretions of the thyroid gland, and nothing else. However, we are entitled to say medically that the established condition may be aggravated by some special strain or stimulus, physical or psychic. These strains may, for instance, precipitate auricular fibrillation.[87] It appears, also, that excessive nervous tension or acute psychic stimuli sometimes act as an effective trigger-mechanism in precipitating the original thyrotoxicosis.[88]

8.a. *Pulmonary heart disease; pulmonary embolism.* Pulmonary heart disease, or the *cor pulmonale,*[89] is uncommon as an important finding. The reader may be helped here if we recapitulate a few of the essential facts covered in our initial discourse on the structure and functions (anatomy and physiology) of the cardiovascular system. It

[84] A low voltage of the *QRS* waves and a very low voltage or even inversion of the *T* waves.

[85] *Thyroid therapy:* This involves administration of dry thyroid gland itself or of equivalent synthetic preparations.

[86] *Metabolism:* The sum of all the physical and chemical processes by which living organized substance is produced and maintained, and also the transformation by which energy is made available for the uses of the organism. An increase in metabolism therefore involves faster and more strenuous functioning by the body and heart and thereby entails an increased work load for the vital organs.

[87] *Auricular fibrillation and flutter:* Are rather pronounced disorders of auricular rhythm (beating of the auricles), possible, however, in the absence of heart disease. Fibrillation results when the excitation impulse which causes contraction of the heart muscles races through the auricle in more or less of a circle ("circus wave"). This causes an incoordinate rapid contraction of the auricles with a very irregular pulse at the wrist before treatment is instituted. *Auricular flutter* is the first cousin of *fibrillation;* it involves a much more regular action at a slower rate and with regular beating of the heart.

[88] *Accord:* SMITH, H. W., AND COBB, S., *Relation of Emotions to Injury and Disease,* 19 ANN. INT. MED. 873 (1943); 30 VA. L. REV. 193 (1944); MITTELMANN, B., *Psychogenic Factors and Psychotherapy in Hyperthyreosis and Rapid Heart Imbalance,* 77 J. NERV. AND MENT. DIS. 455 (1933); MEANS, J. H., THYROID AND ITS DISEASES (1937). For a court case involving alleged reactivation of hyperthyroidism by psychic stimuli, see Walker v. Pitlochry Motor Co., (Scotland, 1930) S. C. 565, (1930) Scot. L. T. 367.

[89] *Cor pulmonale:* Enlargement of the right side of the heart from pulmonary embolism or obstructing pulmonary disease.

will be recalled that after the arterial blood flowing through the capillaries has given up its oxygen to the tissues, and has so become venous blood, it is transmitted by the venules and veins back to the right side of the heart. We saw that it first empties into the right auricle (upper chamber) and at regular intervals, when the right auriculo-ventricular valve opens, the blood flows through the orifice in the floor of the right auricle, into the right ventricle below. At regular intervals the pulmonary valve opens (while the right auriculoventricular valve is closed) and the right ventricle, by contracting, pumps the venous blood into the pulmonary arteries and thence to the vascular bed of the lungs where oxygen is restored by gaseous diffusion through the very thin walls of the minute blood vessels contained in the alveoli or tiny air-sacs of the lung. Next, this blood passes on into larger vessels in the lung and enters the *pulmonary veins* which convey it back to the left auricle (upper chamber of the heart). We saw how, at regular intervals, the mitral valve which separates the left auricle from the left ventricle below opens, allowing the blood to fill up the latter; how the mitral valve then closes synchronously with opening of the aortic valve (separating left ventricle from the aorta). During this time interval (while the right ventricle is contracting to pump venous blood to the lungs), the left ventricle is contracting also, and pumps oxygenated blood out to the body via the aorta, the large artery leading away from the heart.[90]

The reader will now see from the foregoing account that the blood vessels in the lungs constitute a vascular system interposed between the right and the left ventricles of the heart. Any disease or condition which partially or wholly blocks the blood vessels in the lungs will thereby increase the resistance against which the right ventricle of the heart must work in pumping blood to the lungs. This overwork of the right ventricle leads first to hypertrophy (increase in thickness of the muscular wall), and then to dilatation also, and in the end may lead to failure of the heart as a pump. The pulmonary artery may also enlarge as a result of the increased pressure of the blood it carries. Among the lung conditions which may cause varying degrees of pulmonary heart disease are asthma,[91] emphysema,[92] silicosis[93] and pulmonary em-

[90] This chronology of events is called *the cardiac cycle:* it may help the reader to remember that the two auricles contract simultaneously, and a little later the two ventricles; the auriculo-ventricular valves (separating right and left auricles above, from right and left ventricles below) are open when the ventricles are filling with blood; they close when the ventricles contract and pump blood, at which time there occurs an opening of the pulmonary valve (between r. ventricle and pulmonary artery) and the aortic valve (between l. ventricle and the aorta) which had been closed while the ventricles were filling with blood.

[91] *Asthma:* A disease marked by recurrent attacks of paroxysmal dyspnea (sudden labored breathing), with wheezing, coughing, and a sense of constriction, due to spasmodic contraction of the bronchi (air tubes) in the lungs. The paroxysms last from a few minutes to several days, and they may result from direct irrita-

bolism.[94] *Pulmonary heart disease,* or the *cor pulmonale,* is quite rare as an important finding. In slight and negligible degree it is quite frequently found in the course of postmortem examinations, but in such degree it is unrecognizable during life. In high or recognizable degree it can be diagnosed from the finding of enlargement of the right ventricle, and pulmonary artery by X-ray examination[95] and sometimes on physical examination. Electrocardiography is an especially good method of diagnosis in the presence of massive pulmonary embolism (blockage of pulmonary artery by a blood clot carried to it in the blood stream) in acute cases and of extensive pulmonary disease with fibrosis (replacement of normal tissue by fibrous tissue) or emphysema in the chronic cases.[96] *The prognosis* in the acute cases is "touch and go," and in the chronic cases is good sometimes for a considerable number of years. In either case, acute or chronic, it is the pulmonary (lung) involvement rather than the heart disease that is the limiting factor and is responsible for the fatal ending when it comes.

Closely connected with the subject of the acute cor pulmonale and deserving in this paper even more emphasis is *pulmonary embolism*[97]

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tion of the bronchial mucous membrane or from reflex irritation (via the nervous system). Many cases of asthma are allergic manifestations precipitated by the proteins or other substances to which the person has become sensitized.

[92] *Emphysema:* Emphysema is a condition in which the very small air sacs (alveoli) in the lung become distended or ruptured. It may arise from any condition which causes distention of the lungs, particularly those which involve greater interference with expiration than with inspiration, as for instance where a partial obstruction exists in the bronchi (air tubes); asthma, chronic bronchitis and whooping cough are common causes and it may result gradually from such occupations as glass blowing, playing wind instruments or lifting heavy weights regularly. The effect is to rupture some of the alveoli and to cause loss of elasticity in others, the net result being to interfere with exchange of gases in the lungs, and so to impair efficiency of respiration. The onset is slow and gradual and the first evidence is shortness of breath on exertion with perhaps some cyanosis (bluish coloration of the skin).

[93] *Silicosis:* A chronic condition of the lungs caused by inhaling air containing finely divided silica (SO_2) in sufficient quantity and over a long enough period of time to provoke fibrous changes in the alveoli (air sacs). This industrial disease, suspected from a history of such occupational exposure and verified by X-rays of the chest, does not produce symptoms (increased breathlessness on exertion) until its advanced stages. It predisposes to tuberculosis and facilitates the progression of that disease by lowering resistance of the lung tissue.

[94] *Pulmonary embolism:* Blockage of one of the pulmonary arteries by a blood clot brought from another place by the blood stream. See text, *infra* and note 97.

[95] Enlargement of the pulmonary artery involves a dilatation and sometimes a lengthening of the main trunk.

[96] *Technical note:* In the chronic cases the electrocardiogram shows a typical well marked right axis deviation, and in the acute cases it shows prominence of the *S* waves in Lead 1 and of the *Q* waves in Lead 3, lowering of the *T* waves in Lead 2, and inversion of the *T* waves in Lead 3 and often in Lead 4F and in the precordial leads between 1 and 4, that is C_2 and C_3.

[97] *Pulmonary embolism:* An embolus is a blood clot or thrombus that moves from one point to another, either from a vein, most commonly in the leg where the clot is formed, up to the great veins and through the right auricle and right ventricle to block one of the pulmonary arteries, thereby causing what is called pulmonary embolism, or from the inside of the heart in the left auricle or left

itself. Such embolism is quite common, tending to take place after surgical operations, after leg injuries, or after long rest in bed, especially in soft, older persons with sluggish peripheral circulations or a history of known *phlebitis*[98] and most commonly of all in cardiac patients already suffering from congestive heart failure, in whom it is the most common immediate cause of death.[99] It almost always arises from the laying down of a thrombus in the leg veins starting in the calves and usually bilateral (i.e., in both legs); such phlebitis, as it is called, is frequently symptomless and signless and may be discovered only at autopsy or on local exploration by the peripheral vascular surgeon who should be called in to ligate the veins in both legs if they are thought to be the source of trouble in either leg. Such ligation may be life-saving since pulmonary embolism tends to be recurrent and any attack, even a small one, may end fatally, when life already hangs in the balance. After recovery, however, there may be but little residual scarring in the lungs and the patient's health may be excellent.

8.b. *Relation of trauma and strain to pulmonary heart disease and pulmonary embolism.* Pulmonary heart disease in its chronic state may be an occupational disease as in silicosis but it is not caused by trauma or strain although it may be aggravated thereby. On the other hand, acute *cor pulmonale,* dependent as it is on massive acute embolism, frequently can be traced to an injury (trauma) or to an operation which has caused *thrombophlebitis*[100] in the legs with recurrent release of emboli (blood clots) into the blood stream by which they are

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ventricle out into the aorta and thence into one of the peripheral arteries of the body to produce arterial embolism with blocking of the circulation to that particular part of the body, whether leg, brain, or one of the abdominal viscera. This is usually a very serious condition.

Pulmonary embolism is to be diagnosed when there are: (1) Attacks of sudden breathlessness and (2) rapid heart action in an older (indeed, even a younger) person (3) a few days or weeks after an operation or accident, with or without (4) oppressive chest pain, with or without (5) early evidence of the process by X-ray or electrocardiogram, with or without (6) cough and blood spitting, with or without (7) pleurisy pain, and with or without (8) obvious leg phlebitis (inflammation of a vein) which does, however, usually become manifest only after the first attack of pulmonary embolism.

[98] *Phlebitis:* Phlebitis is inflammation of a vein in which there may or may not be deposited a thrombus (blood clot); if it does occur we say the patient has *thrombophlebitis* or *phlebothrombosis.*

[99] See Black Mountain Corp. v. Black, 220 Ky. 85, 294 S. W. 820 (1927) (Death from fatal embolus several weeks after fracture of left femur in coal mine; award of compensation affirmed); Geagan's Case, 301 Mass. 319, 17 N. E. (2d) 172 (1938) (As result of accident X fractured ankle; 6 days later, in hospital, he was operated for an old hydrocele unrelated to injury; 15 days after the operation he died of a pulmonary embolus; medical witnesses disagreed as to whether the embolus resulted from the fracture or from the operation; award of compensation affirmed.).

[100] *Thrombophlebitis:* This involves obstruction of a vein by a blood clot (thrombus) accompanied by an inflammatory reaction in the wall of the vein.

carried to the lungs.[101] Sometimes life may be saved in such cases by ligation (tying off) of the responsible leg veins bilaterally or even of the inferior vena cava, or by the use of heparin (an anticoagulant).[102] The operation of choice is ligation of the common femoral veins bilaterally. It is of more than passing interest that present-day studies are revealing the significant fact that pulmonary embolism (so often fatal) is actually more commonly found in medical patients (especially in cardiacs) than in surgical except for those with leg injuries.

The following clinical description of a case of *pulmonary embolism* is included for *reference purposes* chiefly but should also interest the general reader.

Pulmonary embolism: A. R., white, married, male, 48 years of age, had always enjoyed good health. Ten weeks prior to examination he had pain in his leg while playing tennis. He considered it to be a sprain and treated it accordingly. Four weeks later he had an abdominal pain similar to a stomach ache which radiated up to his left chest and caused pain on respiration for a week. After another 3 and 4 weeks, respectively, he had two attacks characterized by a catch in his throat, pounding of the heart, and rapid difficult respirations. A week after that, he was seen in consultation and his heart and lungs appeared to be normal on auscultation and percussion. The fluoroscopic examination showed a full sized heart with a slightly prominent pulmonary artery.

An electrocardiogram made six weeks after his first attack and five days after his most recent one, showed a sinus tachycardia (rapid heartbeat) at a rate of 125. The S-T segment in Lead 1 originated slightly below the baseline and the S-T segment in Lead 2 made a gradual ascent from below the baseline. There was a small Q wave in Lead 3 and a late and slightly inverted T wave in that lead. There was no abnormal axis deviation.

It was believed that he had a normal heart, but the possibilities of pulmonary embolism, coronary thrombosis, and paroxysmal tachycardia were considered. During the final week he had several spells as previously described and expired in one which was marked by great respiratory distress and cyanosis (bluish coloration of the skin).

At autopsy multiple infarcts were found in both lungs. There was an old laminated clot occluding the main pulmonary artery to the left lower lobe and superimposed on this was a fresh and probably terminal clot. The heart weighed 400 grams and was normal except for some dilatation and hypertrophy (thickening of heart muscle from overwork) of the right ventricle.

[101] See cases cited in note 99 *supra;* also, Sherman v. General Electric Co., 266 App. Div. 1050, 44 N. Y. S. (2d) 661 (1943) (On Sept. 17, 1941, X, in course of work, struck his left knee on the edge of a steel container; traumatic phlebitis resulted; an embolus passed from the leg to the lung causing pulmonary infarction and death on Sept. 17, 1941; held: death was compensable as it resulted from the accidental injury to X's leg sustained on Sept. 17th.).

[102] The reader will recall that the inferior vena cava is a main vein emptying venous blood into the right auricle. Fortunately, adequate connections exist so that when the inferior vena cava is tied off, the venous blood can still reach the right auricle of the heart by way of collateral branches of the vena cava above the point of ligation.

9.a. *Rare kinds of heart disease.* Now we come to the very rare kinds of heart disease, no one of them making up more than a small fraction of one per cent of all cardiac patients. The enlarged inefficient heart of avitaminosis (vitamin deficiency) belongs here, cardiac trauma, and also invasion of myocardium, pericardium, endocardium, or heart chambers by viruses or tumors, primary[103] or metastatic[104] usually undiagnosable during life. Infestation of the heart by trichinosis,[105] trypanosomiasis,[106] and echinococcus[107] is rare in this country. Serious poisoning is uncommon; digitalis is more likely than any other substance to be responsible for the cases one encounters, inducing arrhythmias (irregularities in the beating of the heart) which may be very disturbing though rarely fatal. A notorious instance of the fraudulent use of digitalis to produce apparent heart disease occurred a few years ago in New York City. Several doctors, lawyers, and numbers of insured persons buttressed false claims of heart disease by electrocardiographic abnormalities produced by giving assured excessive doses of digitalis shortly before the heart tracings were made. This fraudulent simulation of heart disease cost insurance companies large sums of money before the swindle was exposed.[108]

9.b. *Relation of trauma and strain to rare kinds of heart disease.* Of the several rare kinds of heart disease mentioned, none save that due to direct cardiac trauma, originates in an injury, but any of them conceivably might be aggravated by an adequate injury, a possibility we shall want to discuss further, *infra,* as it applies to pre-existing impairment of the heart from whatever cause.

10.a. *Relation of trauma and strain to rare kinds of heart disease: traumatic heart disease.* There remains among these lesser causes of structural changes in the heart one that demands further discussion in

[103] *Primary tumor:* The term primary, in respect to a tumor, indicates the site at which it first appeared in the body.

[104] *Metastatic tumor:* Metastatic tumors are secondary tumors caused by the spread of small bits of the tumor mass from the primary site to some other site by way of the blood stream or lymphatic circulation.

[105] *Trichinosis:* Trichinosis is a disease produced by the trichinella spiralis, a kind of worm, particularly as the result of the ingestion of raw or inadequately cooked pork. The encysted larvae are released in the stomach and duodenum, enter the mucous membrane of the gastrointestinal tract, and migrate to the muscles where they become encysted again. The parasites often produce severe pain and fever in the course of this invasion of the muscles of the body.

[106] *Trypanosomiasis:* Trypanosomiasis is an infestation of the body by trypanosomes, resulting from insect bites (Tsetse fly) and certain bugs and often producing a blocking of the lymphatic circulation. It is a cause, for example, of elephantiasis (great enlargement of a part of the body, especially one leg). Trypanosomiasis is a cause of sleeping sickness in Africa.

[107] *Echinococcus:* Echinococcus disease is an infestation of the body by the echinococcus tape worm for which the dog is the primary host and hogs, cattle and man are usually secondary or intermediate hosts; cysts are produced that may invade any structure of the body.

[108] HEDLEY, *The Fraudulent Use of Digitalis to Simulate Heart Disease,* 18 ANN. INT. MED. 154 (1943).

any paper of this type, namely trauma. *Traumatic heart disease* of important degree is rare; very slight injuries may be common, no one knows how common, for they cannot be diagnosed and are of significance only if there is already present a high degree of heart disease due to some other cause. Although attempts have been made to subdivide cardiac injuries according to the tissue involved, that is, myocardium, pericardium, and endocardium, such separation is rather artificial except in the case of some lesions of the heart muscle within the ventricular wall and of some valve ruptures. If the pericardium, or the endocardium lining the heart chambers, is damaged, the subjacent myocardium is also affected and sometimes the injury involves the entire thickness of the heart wall or both pericardium and endocardium with a portion of the underlying myocardium. Damage to the great vessels or to the coronary arteries should be considered separately.

Traumatic heart disease: myocardial contusion (bruising of the heart muscle). Considerable experimental work has been done to determine the ease, the nature, and the extent to which cardiac injury, in particular myocardial contusion (bruising of the heart muscle), is produced by blunt non-penetrating force applied by blows on the thorax (chest) or on the heart itself. Such injury has been difficult to appraise in man in contrast to the relatively simple penetrating wounds. Kulbs (1909),[109] Kulbs and Strauss (1932),[110] Schlomka (1934),[111] Bright and Beck (1935),[112] Kissane, Koons, and Fidler (1936[113] and 1940),[114] and Moritz and Atkins (1938)[115] have demonstrated the effect of blows on the hearts of dogs, cats, and rabbits. Hemorrhagic lesions,[116] small tears, and rupture of the heart walls were commonly produced, and sometimes disorders of function and death resulted with evidence of relatively little actual structural damage. This was shown particularly by Schlomka (1934),[117] who concluded that in experimental animals *commotio cordis* (severe concussion of the heart with disturbance of

[109] KULBS, F., *Experimentelle Untersuchungen über Herz und Trauma,* 19 MITT. A. D. GRENZGEB. D. MED. U. CHIR. 678 (1909).

[110] KULBS, F., AND STRAUSS, L. H., *Herz und Trauma: Weitere experimentelle Untersuchungen,* 11 KLIN. WCHNSCHR. 1572 (1932).

[111] SCHLOMKA, G., *Commotio cordis und ihre Folgen. Die Einwirkung stumpfer Brustwandtraumen auf das Herz.,* 47 ERGEBN. D. INN. MED. U. KINDERH. 1 (1934).

[112] BRIGHT, E. F., AND BECK, C. S., *Non-Penetrating Wounds of the Heart,* 10 AM. HEART J. 293 (1935).

[113] KISSANE, R. W., KOONS, R. A., AND FIDLER, R. S., *Traumatic Rupture of a Normal Aortic Valve,* 12 AM. HEART J. 231 (1936).

[114] KISSANE, R. W., FIDLER, R. S., AND KOONS, R. A., *Traumatic Lesions of the Heart,* Blood, Heart and Circulation, 13 The Science Press 170 (1940).

[115] MORITZ, A. R., AND ATKINS, J. P., *Cardiac Contusion: An Experimental and Pathologic Study,* 24 ARCH. PATH. 445 (1938).

[116] Hemorrhagic lesions are areas of damage, including bleeding, in any tissue of the body.

[117] SCHLOMKA, G., *Experimentelle Untersuchungen uber den Einfluss stumpferbrustkorbtraumen auf das Herz.,* 92 ZTSCHR. F.D. GES. EXPER. MED. 552 (1934).

function caused by shock) due to blows on the heart could result in acute traumatic dilatation of the heart. This is a diagnosis which in man at least, particularly if there is a penetrating injury, must be carefully distinguished from the not uncommon cardiac tamponade[118] brought about by bleeding into the pericardial cavity (the space between the two folds of the pericardial sac which invests the heart) with resultant distention of the sac and increase in size of the X-ray cardiac shadow. Although naturally the right ventricle, being as it is the anterior (forward) heart chamber, receives the brunt of the force from blows on the chest, the actual injuries to myocardium, endocardium, and even pericardium may be widely scattered through the heart.

There still exists a great deal of uncertainty as to how frequently *myocardial* (or *pericardial* (heart sac) and *endocardial* (heart lining)) *contusions* (bruises) and tears occur in man after crushing injuries and blows. Undoubtedly in days gone by such contusions were usually overlooked but nowadays with the lime-light on the heart and on industrial and automobile accident insurance there is a swing of the pendulum in the opposite direction. So far as structural damage is concerned, therefore, the proof of cardiac injury should go beyond mere production of symptoms such as pain, palpitation, or dyspnea (breathlessness). These latter may result from injuries of the chest wall, pleura (membranes investing the lungs), or the lungs, particularly when ribs are broken, or they may be a part of the nervous manifestations of traumatic neurosis.[119] Such proof depends upon finding changes in heart size (by X-ray), abnormal heart sounds or murmurs (by auscultation-listening with a stethoscope) or electrocardiographic anomalies,[120] which were not present before the accident or which may be presumed to have developed acutely following injury in a previously healthy young person (male under 35 years of age, female under 45, older ages being more likely to be attended by electrocardiographic evidence of early or at last symptomless coronary heart disease). It may be noted here, in passing, that no electrocardiographic evidence of any traumatic effect on the heart by boxing was found in a series of 35 young boxers by

[118] *Cardiac tamponade:* Cardiac tamponade is the condition of constriction of the heart by fluid in the pericardium. In the case of trauma, the fluid is usually blood. The pericardial sac, although somewhat elastic, can stretch only to a certain degree. Hence, as the blood pours into the sac, it constricts the heart itself, preventing its normal contraction, and really choking it, resulting in early death.

[119] *Traumatic neurosis:* Neurosis is a disorder of the nervous system, with varied symptoms for which no demonstrable organic basis exists; the symptoms are thought to exist on a psychological basis, representing in many cases, an escape mechanism to get away from a painful situation. Traumatic neurosis follows injury, usually a trivial sort inadequate to produce such disability in an averge person.

[120] Particularly of the *S-T* segments and *T* waves in either of the limb leads or more especially in the precordial leads.

Butterworth and Poindexter (1942).[121] Disorders of function without evidence of structural changes will be discussed later.

Traumatic heart disease: penetrating injuries. Much easier to diagnose and treat, if adequate surgery is close at hand, than cardiac contusions are penetrating injuries, but even these are not common. Elkin[122] has reported that in his own experience only 2 per cent of penetrating wounds of the chest injured the heart, while at the Medical College of Virginia Hospitals, Bigger found that only 0.1 per cent of the surgical patients had such injuries.[123] Elkin presented the findings in 22 cases, mostly of knife wounds, in whom operative cure was attempted; there was a wide variation in the site of the heart lesion, being right ventricular in 9, left ventricular in 5, right auricular in 3, left auricular in 2, aortic in 2, and involving the pulmonary artery in 1. Recovery occurred in 11 cases, including 7 of the 9 with right ventricular penetration, and one each of injury to the left ventricle, the right auricle, the left auricle, and the aorta. The duration of time from injury to admission to hospital when known (in 12 cases) was not over 60 minutes. The causes of death in the fatal cases were as follows: pneumonia in 4, hemorrhage in 3, and pericarditis (inflammation of the pericardium, the membranous sac which contains the heart), bacteriemia (invasion of the blood stream by bacteria), "infection," and mediastinal emphysema[124] in 1 each. Without surgery these cases presumably would all have died from *cardiac tamponade,* which involves compression of the heart by effusion of blood into the pericardial sac. The effect of this is to prevent blood from entering the heart from the great veins and as a result the output of the heart falls almost to nil (the venous pressure rises and the arterial pressure falls). For *reference purposes,* it seems desirable to summarize several case histories of traumatic heart disease, but the general reader may pass over these without any disturbance to the continuity of the discourse.

Traumatic heart disease: Case 1: Immediately Fatal Injury to Normal Heart from Automobile Passing Over Chest, Without Evidence of

[121] BUTTERWORTH, J. S., AND POINDEXTER, C. A., *An Electrocardiographic Study of the Effects of Boxing,* 23 AM. HEART J. 59 (1942).

[122] ELKIN, D. C., *The Diagnosis and Treatment of Wounds of the Heart, A Review of Twenty-Two Cases,* 111 A. M. A. JOUR. 1750 (1938).

[123] BIGGER, I. A., *Heart Wounds, A Report of Seventeen Patients Operated On in the Medical College of Virginia Hospital, and Discussion of Treatment and Prognosis,* 8 J. THORACIC SURG. 239 (1939).

[124] *Mediastinal emphysema:* The abnormal presence of air in the mediastinal tissue. The mediastinum is the space between the two pleural sacs investing the right and left lungs; it extends from the sternum (breast bone) in front to the thoracic vertebrae (backbone) behind and from the thoracic inlet (level of the collar bones) above to the diaphragm below (the diaphragm is the sheetlike muscle which separates the chest cavity from the abdominal cavity.

External Injury. Hamilton[125] reports the case of a girl, aged seven years, who was brought to the Cumberland Infirmary following an accident, in which the front wheel of a car had passed over her chest. External examination revealed a slight abrasion (spot rubbed bare of skin) on the forehead, but nothing more.

At postmortem examination all the ribs were found to be intact; the left pleural cavity (space between the two layers of the pleural sac which invests the lung) contained approximately 24 ounces of blood; the pericardium (heart sac) was split longitudinally on the left side and both ventricles were ruptured, the right one to a greater extent than the left.

Traumatic heart disease: Case 2: Rupture of the Right Ventricle from Abdominal Compression with Hemopericardium[126] *and Death Four Months Following the Injury.* Schorre reports the case of a man, aged fifty-six years, previously healthy and apparently free from hypertension (high blood pressure), who fell with his abdomen against one of the horizontal bars on which he was exercising, on August 8, 1933. He continued to exercise that day, but the next day had pain in his abdomen; after five days he found walking difficult because of shortness of breath, which constantly grew worse. Otherwise, he felt well. Physical examination showed no abnormalities in abdomen or lungs. The heart was regular, with soft sounds, but with much increased percussion dulness in all directions, confirmed by X-ray. His blood-pressure then measured 195 systolic and 90 diastolic, but it later dropped to 115 systolic and 85 diastolic. The symptoms increasing, a diagnosis of pericardial effusion (leakage of blood into the space between the two layers of the heart sac) was made, and on September 22, 1933, five weeks after his injury, a pericardial tap[127] yielded 200 c.c. of blood. A second tap, on October 10, again yielded 200 c.c. of blood. The heart dulness distinctly decreased. After temporary improvement, his condition became worse again. The apex-beat could not be felt, and the heart sounds were hardly audible at the apex. Slight cyanosis of the lips and hands developed, the pulse became paradoxical,[128] dyspnea increased, and he died on December 12, 1933, about four months after the accident.

Autopsy showed a canal-like rupture of the right ventricular wall which made a circuitous path in the region of the infundibulum.[129] The wall of the right ventricle was considerably infiltrated with fat, especially in the region of the rupture. The coronary arteries were not narrowed. The aorta was not dilated, nor very sclerotic. The heart muscle showed no scars but some softening and thinning in the region of the rupture.

[125] HAMILTON, J. A., *Traumatic Rupture of the Heart Without External Injuries,* 2 BRIT. MED. J. 1101 (1934).

[126] *Hemopericardium:* Hemopericardium is the collection of blood in the pericardium due to tuberculosis, malignancy, or trauma.

[127] SCHORRE, E., *Über Herzrupturen nach Bauchtraumen,* 27 ZTSCHR. F. KREISLAUFFORSCH. 577 (1935).

[128] *Paradoxical pulse:* Paradoxical pulse is an unusual change in the form of the pulse in which the pulse becomes smaller on inspiration rather than larger and may even disappear entirely for a beat or two.

[129] *Infundibulum:* The infundibulum of the right ventricle is the part of the ventricle in the region of the outflow tract just proximal to the pulmonary valve and artery.

The pericardium showed some fibrosis microscopically, with round-cell infiltration,[130] but no tubercles.[131] There was a high degree of stasis[132] in the abdominal organs.

Traumatic heart disease: Case 3: Injury to a Relatively Normal Heart from a Blow on the Chest (Steering-wheel Type of Injury) with Death Six Days After the Accident from Congestive Heart Failure. Beck[133] reports the following case: A man, aged forty-nine years, was injured by driving his car into the cement buttress of a bridge. He was thrown forward with his chest against the steering wheel. He extricated himself from the wreck, got out and walked around the car. He wanted to continue his journey, but was persuaded to go to a hotel. He sustained fractures of several ribs. These were strapped and, against his wishes, he was placed in a hospital. He said he felt all right except for some pain from the fractures. On the third day after the accident he had a rapid pulse and seemed to be seriously ill. On the fourth day, he showed a tinge of cyanosis, slight dyspnea, and a pulse-rate of 136 per minute. Frequent premature ventricular contractions were present. The electrocardiograms showed evidence of myocardial (heart muscle) injury. The heart sounds were reduced in intensity and had a peculiar "tick-tock" quality. The sounds were similar to those heard by Beck in his experimental cardiac contusions, and this peculiar quality helped him to make a diagnosis of cardiac contusion. The fourth, fifth, and sixth ribs on the left were fractured. The sternum was slightly depressed and had a transverse fracture line in its lower third. There was no evidence of hemothorax,[134] pneumothorax,[135] pneumonia, or abdominal complication. On the basis of a diagnosis of myocardial contusion (bruising of the heart muscle), complete bed rest was advised. Six days after the injury, death occurred from myocardial failure. The necropsy[136] showed two contusions in the posterior wall of the right ventricle the size of a dime (18 mm.), and a laceration (tear or cut) of the myocardium between these areas. The heart had not ruptured through the whole thickness of the heart wall. There were found some coronary sclerosis (arteriosclerosis of the coronary artery) and some fatty infiltration of the heart.

Traumatic heart disease: Case 4: Injury to Previously Normal Heart, with Survival. Bullet Lodged in Myocardium (Heart Muscle).

[130] *Round cell infiltration:* Round cell infiltration consists of a collection of round cells (a type of white blood cell) in some tissue of the body due to some particular, often localized, reaction.

[131] *Tubercles:* Tubercles are masses of cells commonly representative of tuberculosis, including round cells and giant cells, due to local reaction of the body to the presence of infections or toxic agents. They vary in size from microscopic specks to masses that are easily visible.

[132] *Stasis:* Stasis usually refers to a sluggish course of the blood stream in some part of the body, or even to a standstill of the circulation.

[133] BECK, C. S., *Wounds of Heart; The Technique of Suture,* 13 ARCH. SURG. 205 (1926).

[134] *Hemothorax:* Hemothorax is a collection of blood in the pleural cavity (i.e., between the two layers of the pleural sac which invests each lung) due to injury or infection.

[135] *Pneumothorax:* Pneumothorax consists of a collection of air in the pleural cavity due to trauma or infection.

[136] *Necropsy:* Necropsy is a synonym for autopsy or postmortem examination.

B. F., aged sixteen years, shot himself accidentally while hunting, on June 24, 1935. He was immediately brought to the hospital, complaining of pain in the left shoulder, and was found to be in mild shock. The temperature was 100.2°F. by rectum, and the pulse, which was 84 on admission to the hospital, increased to 120 in the course of a few hours. The blood-pressure was 110 systolic and 70 diastolic; the respirations were 20. There was a bullet wound about 1 cm. below the left costal margin, in the midclavicular line. The bullet tract was directed upward and slightly toward the midline. The heart showed no apparent enlargement. The rhythm was regular and the sounds were of good quality. Examination of the blood revealed: red blood cells, 4,250,000;[137] hemoglobin,[138] 80 per cent; white blood cells,[139] 9,050, with 84 per cent polymorphonuclears.[140] The urine was negative.

After one week the patient's temperature varied from 98.6° to 100.2°F., and the pulse from 90 to 120. Following this, both temperature and pulse became normal and have remained so; the blood-pressure rose to 120 systolic and 80 diastolic. He had had no complaints.

On July 1, 1935, the seventh day after the injury, fluoroscopic examination and X-ray films revealed a bullet in the lower left portion of the heart shadow, posteriorly, about 1 inch above the diaphragm; this moved with the heart-beat; during systole it moved laterally; it did not change its location when the patient was tilted upright on the table.

Electrocardiograms were characteristic of those seen in myocardial infarction of the posterior or diaphragmatic type.

The patient was given absolute bed rest, and all operative procedures were discouraged. Convalescence was uneventful. Four and a half months after his injury, his recovery seemed nearly complete, and he was leading a normal life. Dr. E. O. Black, of Fredonia, N. Y., reports that five years after the injury the patient was well and pursuing his usual activities.

Traumatic heart disease: Case 5: Injury to Previously Normal Heart, with Survival. Stab-wound Severing a Coronary Vessel and Injuring the Myocardium (Heart Muscle). Successful Ligation and Suture. Bigger and Porter[141] report the case of a colored man aged twenty-three years, who was admitted to the St. Phillip Hospital, on October 2, 1930, at 12 noon. He had been stabbed with a knife in the

[137] *Red blood cell count:* The normal red blood cell count varies from 4,000,000 to 5,000,000 red blood cells per cubic millimeter. Hence, 4,500,000 is within the normal range.

[138] *Hemoglobin:* Hemoglobin refers to the amount of the iron-protein content in the blood capable of absorbing oxygen to transport it to the lungs and tissues. Normally, its range is from 75 to 100 per cent as measured by a certain scale called the Sahli or Tallquist, but its amount is also indicated as actual weight of the hemoglobin in a certain amount of blood, the normal weight varying from 13 to 15 grams per 100 cubic centimeters of blood.

[139] *White blood cell count:* The normal white blood count averages 8,000 per cubic millimeter, ranging from 6,000 to 10,000.

[140] *Polymorphonuclear:* Polymorphonuclear refers to white blood cells with multilobed nuclei. These are the cells that are called into action in infectious disease or trauma. Normally the percentage of the white blood cells that are polymorphonuclear in type is 60 to 70.

[141] BIGGER, I. A., AND PORTER, W. B., *Wounds of the Heart,* 1 INTERNAT. CLIN. (Series 44) 132 (1934).

left side of the chest approximately thirty minutes before admission. When admitted to the hospital, the patient appeared to be in profound shock; his radial pulse[142] was not palpable, and his blood-pressure could not be obtained. Examination revealed a wound in the fourth intercostal space (space between the 4th and 5th ribs), 5 cm. to the left of the sternum (breast bone). The heart sounds were distant and muffled, and the signs of shock were out of proportion to the external blood loss; the diagnosis of heart wound with heart tamponade was, therefore, made and immediate operation advised.

A penetrating wound of the anterior wall of the left ventricle was found, which had divided the descending branch of the left coronary artery. The vessel was ligated, and the wound in the left ventricle repaired with interrupted sutures of fine silk.

The patient made a good recovery, and left the hospital sixteen days after the operation.

Case 6: Non-penetrating Wound of the Heart.[143] *Rupture of Papillary Muscle*[144] *and Contusion*[145] *of Heart, Resulting from External Violence, without Definite Clinical Evidence of Injury to the Heart. Death from Multiple Complications.* Glendy and White report the case of a seaman, aged twenty-four years, who had been in good health up to the time of his injury, which occurred on August 7, 1933. On that date, while asleep and riding in an automobile driven by a shipmate, an accident occurred; the patient was thrown violently from the car, and a truck ran over his upper abdomen. He was taken immediately to a Relief Station, complaining of pain in the left kidney region posteriorly, radiating to the flank. There, a diagnosis was made of contusion of the abdomen, with possible rupture of the spleen or left kidney. He was given first-aid treatment and transferred by ambulance to a hospital.

On admission to the hospital, the patient was conscious, but apparently suffering from hemorrhage and shock. The skin was cold and pale, the pulse was rapid and weak; he was still complaining of severe pain in the left kidney region posteriorly, and of severe headache. Examination revealed small lacerations and contusions over the scalp and body. Chest expansion was limited bilaterally by pain in the left flank. Anteriorly, there were no abnormal signs over the lungs. The back of the chest was not examined. There was no apparent cardiac enlargement. The heart action was regular at a rate of 88, the sounds were of good quality, and there were no murmurs. The blood-pressure was 100 systolic and 70 diastolic. The entire abdomen was tense. There was board-like rigidity and tenderness in the left upper quadrant.[146] X-ray

[142] *Radial pulse:* The radial pulse is the arterial pulse felt at the wrist close to the end of the radius bone.

[143] GLENDY, R. E., AND WHITE, P. D., *Nonpenetrating Wound of Heart: Rupture of Papillary Muscle and Contusion of Heart Resulting from External Violence. Case Report.* 11 AM. HEART J. 366 (1936).

[144] *Papillary muscle:* The papillary muscle of the heart is a mass of muscle extending into the cavity of either right or left ventricle from the wall to support the chordae tendineae, the fine tough strands which hold the valve cusps in place (see Figure 1).

[145] *Contusion:* Contusion of the heart involves bruising of the heart by direct or indirect trauma.

[146] *Left upper quadrant:* The left upper quadrant of the abdomen is the upper left quarter lying under the left rib edge and to the left of the midline.

films showed a fracture of the eleventh rib on the left. There was no evidence of lung collapse, or of any extensive pathological change within the chest. The urine was grossly bloody. The hemoglobin was 60 per cent (Tallquist);[147] the red blood count, 2,740,000; the white blood count, 21,000, with 84 per cent polymorphonuclears.

After a transfusion of 500 c. c. of whole blood, an exploratory laparotomy[148] was done. This revealed a ruptured spleen which was removed. The patient's condition was never satisfactory after the operation, and in spite of intravenous injections of fluid, digitalis therapy, and the administration of oxygen, he grew steadily worse. His temperature rose to 107.4°F., and he died on August 9, 1933, about twenty-six hours following his injury. Death resulted from loss of blood, shock, and an overwhelming pneumonic infection following the removal of the ruptured spleen.

At autopsy, the anterior papillary muscle in the left ventricle was found to be ruptured at its base, producing a hemorrhagic lesion (2 by 2.5 cm. wide and 1 cm. deep) within the myocardium. Overlying this area on the external surface of the myocardium was a purplish contusion, 3 by 1.5 cm. in area.

Traumatic heart disease: Possibility of erroneously imputing symptoms to heart injury when true cause is an independent coronary thrombosis. When the myocardium is bruised (contused) or torn and the victim survives for hours, days, or weeks, the clinical evidence is much the same as that produced by myocardial infarction (pain, fever, leukocytosis,[149] electrocardiographic changes, and if the lesion is very large, congestive heart failure). In fact, histologically,[150] except for more hemorrhage and tearing of fibers in acute traumatic cases and a fresh thrombus in a coronary artery in the medically diseased cases, it may be impossible to distinguish the myocardial lesions due to injury from those due to acute coronary thrombosis after the process of healing has set in.[151] It is to be remembered that an acute myocardial infarct[152] due to coronary thrombosis may rupture in the first week and so a muscle tear is not adequate evidence either. Naturally, after the tying of a traumatically incised or punctured coronary artery, the microscopic

[147] *Tallquist:* Tallquist is the name of the man who introduced one of the methods for determining the amount of hemoglobin in the blood by color index. He constructed a color scale against which a fresh blood stain is read directly.

[148] *Laparotomy:* Laparotomy consists of a surgical incision into the abdominal cavity.

[149] *Leukocytosis:* An increase in the number of white blood cells; this occurs normally during digestion and in pregnancy, but also occurs as a blood response to infection, and various other disease processes.

[150] *Histological:* Histological refers to microscopic examination of tissue.

[151] MORITZ, A. R., AND ATKINS, J. P., *Cardiac Contusion: An Experimental and Pathologic Study,* 25 Arch. Path. 445 (1938).

[152] *Myocardial infarct:* A myocardial infarct consists of damage to and destruction of a variable amount of the heart muscle due to cutting off of its blood supply. "Coronary thrombosis" means that a thrombus or blood clot has plugged up the coronary artery: this of course stops or greatly reduces the blood flow through the artery.

differentiation of the resultant infarct from that due to *spontaneous thrombosis* of that artery is impossible.

Traumatic heart disease: Rupture of heart valves. Rupture of heart valves by trauma as from crushing, blows, falls, or muscular strain is very rare, especially when the valves are normal to start with but there are authentic cases of such injuries (Barié, 1881,[153] Kissane *et al.*, 1936,[154] and Adam, 1937).[155] The aortic valve (which guards the orifice between the left ventricle and the aorta) is much more often ruptured than the others. When the mitral valve is damaged it is usually through a break of the *chordae tendineae*.[156] Again for *reference purposes,* rather than for present perusal by the general reader, we are presenting medical case reports illustrating occurrence of valve ruptures without previous disease of the valves.

Traumatic heart disease: Case 7: Rupture, by Strain, of a Normal Aortic Valve, with Immediate Onset of Symptoms, and Death Twelve Weeks Later from Progressive Congestive Heart Failure. Howard[157] reports in detail the case of a man, aged thirty-three years, formerly in good health, who entered the Montreal General Hospital complaining of pain over the heart, palpitation, shortness of breath, cough, and swelling of the feet. One month previously, on a cold morning, after cranking his car for two minutes, the patient was suddenly seized with a sharp pain in the left upper chest, and especially in the epigastrium,[158] but with no radiation to the arm;[159] he immediately became short of breath and began to cough. He remained at work for several hours, when he stopped because of the pain in the epigastrium and dyspnea (difficult breathing). Subsequently he noticed a "thrill"[160] in the upper chest. He remained in bed for two weeks and then returned to work; but six days before his admission to the hospital he was forced to discontinue work, as his symptoms had become very acute. The pain at that time radiated down the left arm and even to the left leg. He became very orthopneic[161] and vomited constantly. Edema (swelling) of the feet also appeared.

[153] BARIÉ, E., *Recherches cliniques et experimentales sur les ruptures valvulaires du coeur*, 1 REV. DE MED. DE PARIS 132, 309 (1881).

[154] KISSANE, R. W., KOONS, R. A., AND FIDLER, R. S., *Traumatic Rupture of a Normal Aortic Valve*, 12 AM. HEART J. 231 (1936).

[155] ADAM, A., *Uber die traumatischen Veranderungen gesunder Klappen des Herzens*, 19 ZTSCHR. F. KREISLAUFFORSCH. 313 (1927).

[156] *Chordae tendineae:* The chordae tendineae are the strong fibrous strands attaching the edges of the auriculoventricular valve cusps to the papillary muscles (see Figure 1).

[157] HOWARD, C. P., *Aortic Insufficiency Due to Rupture by Strain of a Normal Aortic Valve*, 19 CANAD. MED. ASSN. JOUR. 12 (1928).

[158] *Epigastrium:* The epigastrium is the upper part of the abdomen in the midline overlying the stomach itself.

[159] *Radiation:* Radiation to the arm refers to the spread of pain or numbness into and down the arm.

[160] *Thrill:* A thrill is a palpable vibration over the heart or lungs.

[161] *Orthopnea:* Orthopnea is a state of difficult breathing or dyspnea in which it is impossible or difficult for the patient to lie flat or even at some angle. It is necessary for the individual to breathe sitting or standing in the upright position.

Physical examination revealed a large man, in great respiratory distress. The color of the face was sallow, but cyanosis (bluish coloration) of the lips, ears, and fingertips was present. There were slight dulness, diminished breath sounds, and many moist râles at both lung bases. There was a slight bulging of the precordium,[162] a widespread heaving impulse was visible over the entire left chest, with its maximum intensity in the fifth interspace, 12 cm. from the midsternum. An intense vibratory thrill could be felt at the base of the heart. It was perceptible throughout the entire cardiac cycle, but with definite systolic and diastolic accentuation. On auscultation,[163] the heart sounds were replaced by murmurs, whose maximum intensity was at the third and fourth left interspaces, close to the sternum. A rough murmur, filling the whole systolic period and obliterating the first sound, was widely distributed, but of maximum intensity in the second right interspace. A high-pitched diastolic murmur, also widely transmitted, was heard with maximum intensity along the left sternal border. No second sound could be heard. The pulse was of the typical water-hammer character and was rapid, with a palpable vibratory thrill. The blood-pressure was 130 systolic and from 40 to 0 diastolic. There was marked pulsation of all the peripheral vessels. The electrocardiogram showed an inversion of the *T* wave in all leads. Fluoroscopy revealed an enormous heart with no aortic aneurysm. The liver was enlarged and tender. The legs were edematous. There was a constant albuminuria.[164] The blood count was normal (hemoglobin, 75 per cent; red blood cells, 5,080,000) except for an occasional leukocytosis (7,500 to 11,000). The Wassermann test[165] was negative.

The diagnosis favored was either a congenital heart lesion with cardiac decompensation,[166] or an acquired aortic insufficiency[167] with the sudden onset of cardiac decompensation due to strain; the possibility of a rupture of an aortic cusp,[168] however, was suggested and seriously considered.

The course was a downhill one, in spite of short periods of improvement due to bed rest and digitalis. Death occurred from progressive cardiac failure, twelve weeks after the severe muscular effort.

Postmortem Examination. The common attachment of the anterior and medial (left posterior) cusps of the aortic valve was torn away from the aortic wall, due to a transverse tear in the intima,[169] 5/8 inch in length, which allowed the cusps to become very lax. The mitral

[162] *Precordium:* The precordium is that part of the anterior chest wall overlying the heart.

[163] *Auscultation:* Auscultation is a method of examining the heart and lungs which involves listening to the heart or breath sounds with a stethoscope.

[164] *Albuminuria:* Albuminuria is the presence of albumin in the urine, an abnormal state.

[165] *Wassermann test:* The Wassermann test is a serological reaction of the blood used as an aid in determining whether or not a given person has syphilis.

[166] *Cardiac decompensation:* Cardiac decompensation is failure of the heart muscle to carry on the normal circulation resulting in congestion of the lungs or of the systemic circulation, that is, of the body in general.

[167] *Aortic insufficiency:* Aortic insufficiency consists of leakage of blood through the aortic valve back into the left ventricle from the aorta.

[168] *Aortic cusp:* An aortic cusp is one of the three small curtains making up the aortic valve.

[169] *Intima:* The intima is the inner lining of a blood vessel.

valve showed slight thickening. There was a fairly extensive sclerosis in the sinus of Valsalva,[170] and an earlier process in the root and arch of the aorta, as well as in the descending aorta. The heart weighed 640 grams, and showed marked hypertrophy[171] with much dilatation of the left ventricle and great dilatation of the right. The other organs revealed marked passive congestion.[172] There were multiple lung infarcts. Microscopically the aortic cusp was normal. The vasa vasorum[173] of the aorta showed no evidence of syphilis.

Traumatic heart disease: Case 8: Rupture of Normal Chordae Tendineae of the Mitral Valve, Resulting in Incompetency of the Mitral Valve and Death from Congestive Heart Failure Eight Months Later. Frothingham and Haas[174] report the case of a man, aged sixty-five years, previously in good health, who rowed and swam strenuously during the course of an afternoon; that night he had pulmonary edema and tachycardia.[175] Congestive heart failure gradually developed, the patient never responding well to adequate treatment; he died after eight months, the immediate cause of death being bronchopneumonia. Autopsy revealed rupture of four undiseased chordae tendineae of the posterior cusp of the mitral valve. The right auricle and right ventricle were greatly distended. The left auricle was much less prominent than the right. The left ventricle was relatively contracted. The heart weighed 360 grams. The heart muscle appeared normal on gross and microscopic examination.

The authors speculated as to the relationship between the strenuous activity and the ruptured chordae tendineae. It seemed reasonable to assume that the onset of the cardiac symptoms was associated with the rupture of the chordae tendineae and the subsequent incompetency of the mitral valve. Just when the rupture occurred was not certain. It would be natural for the rupture to occur during the exertion itself. But since there was an interval of several hours following the exertion, during which the patient was symptom-free, it was suggested that one or more of the chordae might have ruptured in the afternoon, at the time of the exertion, and that this rupture imposed an unusual strain on the remaining chordae which ruptured later, resulting in incompetency of the mitral valve, and marking the onset of the symptoms. The authors point out the interesting fact that prolonged cardiac decompensation can result from pure dilatation of the heart, with little or no increase in the weight of the heart.

Rupture of diseased heart valves without trauma as the cause is, on

[170] *Sinus of Valsalva:* A sinus of Valsalva is the cavity at the beginning of the aorta behind each of the valve cusps.

[171] *Hypertrophy:* Hypertrophy consists of enlargement, usually from overwork. In the case of the heart it involves the muscle or myocardium.

[172] *Passive congestion:* Passive congestion is congestion due to the accumulation of blood in the tissue, not the result of infection. It is characteristic of congestive heart failure.

[173] *Vasa vasorum:* The vasa vasorum are the minute blood vessels of the aortic wall which maintain its nutrition. They mean strictly blood vessels of the blood vessels.

[174] FROTHINGHAM, C., AND HAAS, G. M., *Rupture of Normal Chordae Tendineae of the Mitral Valve,* 2 AM. HEART J. 492 (1934).

[175] *Tachycardia:* Tachycardia means rapid heart rate.

the other hand, not very rare, especially in cases of subacute bacterial endocarditis where there may be considerable destruction of the valve structure by the infectious process. Also eversion of an aortic cusp in syphilitic or even in rheumatic heart disease may give rise to an unusual aortic diastolic murmur. Finally, under any condition, valve rupture is a serious strain on the heart leading as a rule to heart muscle failure and often to early death.

Traumatic injury of the aorta. The aorta may rupture also as the result of blows or falls, even when normal, as has been pointed out by Copeland (1914)[176] and Collins and D'Alessio (1938)[177] but *spontaneous rupture* of diseased aortas is infinitely more common, as in the case of syphilitic and even arteriosclerotic aneurysms,[178] and dissection of weak aortic walls in cases of *medionecrosis aortae* (dissecting aortic aneurysms).[179] The most common traumatic injury of the aorta is by penetration (bullet, shrapnel, knife); this is amenable to cure by suture (sewing) if surgical help is quickly available.

Traumatic injury of the other great blood vessels. The other great vessels, the pulmonary artery,[180] the venae cavae,[181] and branches of the thoracic aorta[182] are injured but infrequently and then in the main only by penetrating wounds. Injury of the coronary arteries similarly may result in hemopericardium with cardiac tamponade. The treatment is to tie off the vessel involved and this in turn results in myocardial infarction, as the effect is to deprive some of the heart tissue of its blood supply.[183]

[176] COPELAND, G. G., *Traumatic Rupture of the Healthy Aorta without External Signs of the Cause of Death*, 63 A. M. A. JOUR. 1950 (1914).

[177] COLLINS, J. O., AND D'ALESSIO, C. M., *Traumatic Rupture of the Thoracic Aorta*, 219 NEW ENGLAND JOUR. MED. 229 (1938).

[178] *Arteriosclerotic aneurysms:* Arteriosclerosis and high blood pressure, in the absence of syphilis frequently produce diffuse dilatation of the aorta but only rarely an aneurysm. Sometimes it does do so in the manner described in note 179, *infra*.

[179] *Dissecting aortic aneurysm:* A dissecting aortic aneurysm consists of the splitting of the wall of the aorta. Blood penetrates to produce a bulging and eventually a rupture of the outer part of the wall, with death from hemorrhage. High blood pressure is often a contributing factor. It is characterized by sudden agonizing pain in the chest (thorax) or abdomen developing, often, during great physical exertion and followed by shock. Death usually occurs within a few hours or days from rupture of the aneurysm. Absence of electrocardiographic changes helps to differentiate this condition from coronary occlusion which produces similar symptoms.

[180] *The pulmonary artery:* The pulmonary artery is the large artery leading from the right ventricle to the lungs (see Figure 1).

[181] *The venae cavae:* The venae cavae are the two large veins bringing blood into the right auricle, the superior vena cava from the upper part of the body and the inferior vena cava from the lower part of the body (see Figure 1).

[182] *The thoracic aorta:* The major branches of the thoracic aorta consist of three major trunks, the innominate which comes off to the right and divides into the right subclavian and the right carotid, the left carotid, and the left subclavian arteries (see Figure 1).

[183] *See* Diehl v. General Baking Co., 349 Pa. 235, 36 A. (2d) 801 (1944) (rup-

FUNCTIONAL DISORDERS OF THE CIRCULATION AND THEIR MEDICOLEGAL ASPECTS ALSO NEED TO BE BRIEFLY CONSIDERED

Introductory remarks. Functional disorders of the circulation may or may not be associated with structural abnormalities or evident etiological factors. The gravest functional disorders of the heart, namely: congestive heart failure, coronary insufficiency, ventricular paroxysmal tachycardia,[184] and high grade heart block, are almost invariably complications of serious heart disease. Generally such heart disease is of long standing but occasionally it is of recent and sudden origin as in cases of acute coronary thrombosis[185] and of acute rheumatic myocarditis.[186] Lesser disorders of the heart such as certain arrhythmias, neurocirculatory asthenia,[187] and cardiac neurosis[188] need brief mention, also. Finally, *peripheral vascular failure*[189] may be transient and unimportant as in simple syncope,[190] or it may be very serious, threatening life, as in the case of vascular shock. Shock practically always accompanies some obviously grave condition such as hemorrhage, post-operative collapse, or accident. The primary condition may or may not

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ture of aortic aneurysm); Keely v. Metropolitan Edison Co., 157 Pa. Super. 63, 41 A. (2d) 420 (1944) (steering wheel impact, when car overturned, caused a tear an inch long in a normal aorta, as proven by postmortem examination after employee died; for a week he went about his work after the injury complaining of pain before he collapsed and died; autopsy showed that after the aortic wall was torn, the blood dissected between its layers and finally entered the pericardial sac, mechanically stopping the heart (cardiac tamponade)).

[184] *Ventricular paroxysmal tachycardia:* Ventricular paroxysmal tachycardia is an attack of very rapid action of the heart initiated by irritabiilty in the ventricular muscle; it is usually a serious state.

[185] *Acute coronary thrombosis:* Sudden blockage of a coronary artery by a blood clot resulting in death of the heart muscle in the area supplied by the occluded vessel. The initial symptom is usually severe substernal pain (behind the breast bone). Unlike the pain of angina pectoris, that of coronary thrombosis is usually spontaneous, occurring at rest more often than after exertion and it is not abolished by rest but frequently persists for hours, not being relieved by nitrites as is angina pectoris.

[186] *Acute rheumatic myocarditis:* Severe inflammatory disease of heart muscle resulting from rheumatic fever.

[187] *Neurocirculatory asthenia:* Neurocirculatory asthenia is a syndrome of physical unfitness of unknown origin, consisting of a collection of symptoms without evidence of structural disease. The symptoms include dyspnea with sighing respiration, palpitation, precordial aches and pains, nervousness, easy exhaustion, tremor, increased perspiration, and a tendency to faint (see later discussion under Neurocirculatory Asthenia).

[188] *Cardiac neurosis:* Cardiac neurosis is a psychoneurosis with anxiety about the heart with, or without, any symptoms otherwise. Usually there is no demonstrable organic heart disease present. (See later discussion under Cardiac Neurosis).

[189] *Peripheral vascular failure:* Peripheral vascular failure consists of failure of the blood vessels of the body through lack of proper action by way of constriction. Usually the blood is pooled in the blood vessels and there is inadequate circulation of blood to the brain, coronary arteries, and other vital structures.

[190] *Syncope:* Syncope is fainting; it involves temporary unconsciousness from a suddenly diminished blood supply to the brain (cerebral anemia). One aspect of treatment is to lower the patient's head and to elevate his extremities.

involve the heart or aorta or cause hemorrhage, acute myocardial infarction, dissecting aortic aneurysm, and pulmonary or peripheral arterial embolism. Such shock is manifested by prostration, a marked drop in blood pressure, a gray pallor, and a threat of death. After available measures have been taken to control the exciting cause, such as hemorrhage, the one specific treatment is repeated blood transfusion.[191]

Functional disorders of the circulation (continued) : *Congestive heart failure.* Congestive heart failure (myocardial insufficiency or failure) is a common sequel of severe prolonged strain on the heart muscle from any cause, most commonly due to high blood pressure and valvular deformity (from rheumatic, syphilitic, or calcareous valvular heart disease). Myocardial infarction (death of heart tissue) due to coronary occlusion (closure of coronary artery) and acute rheumatic myocarditis (inflammation of heart muscle caused by acute rheumatic fever) are other fairly common causes. Other etiological factors are rare, and include trauma, very rapid paroxysmal tachycardia (highly accelerated heart rate) and auricular fibrillation,[192] pulmonary disease, congenital defects, and a few miscellaneous conditions mentioned earlier in this study as being among the rarest kinds of heart disease. Congestive heart failure most commonly involves the left heart chambers, in particular the left ventricle, since the strains on that side (imposed by essential hypertension,[193] aortic valve disease,[194] and myocardial infarction)[195] are far more numerous and greater, by at least 3 or 4 to 1, than the strains on the right heart chambers (imposed by mitral stenosis,[196] extensive pul-

[191] *See* E. Baggot Co. v. Industrial Commission, 290 Ill. 530, 125 N. E. 254, 7 A. L. R. 1611 (1919) (Hemorrhage from ruptured aorta) ; State v. District Court of Brown County, 137 Minn. 30, 162 N. W. 678 (1917) (Ruptured blood vessel) ; Murray v. Cummings Const. Co., 232 N. Y. 507, 134 N. E. 549 (1921) (Cerebral hemorrhage) ; Clark v. Lehigh Valley Coal Co., 264 Pa. 529, 107 Atl. 858 (1919) (Rupture of aorta caused by ". . . an extra effort in vomiting") ; Southwestern Surety Ins. Co. v. Owens, 198 S. W. 662 (Tex. Civ. App. 1917) (Rupture of blood vessel in lungs allegedly due to lifting).

[192] *Auricular fibrillation:* The individual heart fibers take up their own independent action, producing an incoordinate or vermicular contraction of the auricles; as a result the heart beats and the pulse beats become extremely irregular both as to time and as to force.

[193] *Essential hypertension:* Essential hypertension is high blood pressure of unknown cause which strains the left side of the heart by great increase in its work.

[194] *Aortic valve disease:* Aortic valve disease causes damage to the aortic valve thus producing either leakage or stenosis, that is, narrowing, which increases greatly the work of the left ventricle.

[195] *Myocardial infarction:* Myocardial infarction consists of fresh or old scarring of the heart muscle, cutting out of action a variable amount of the muscle, and thereby often increasing greatly the work of the remaining ventricular muscle.

[196] *Mitral stenosis:* Mitral stenosis acts to produce strain on the right side of the heart mechanically because blood cannot be easily forced through the narrow opening which now exists between the left auricle and the left ventricle.

monary disease,[197] and congenital defects).[198] Left ventricular failure, followed at once by left auricular failure, results in congestion of the lung circulation, sometimes acute (acute pulmonary edema, with or without cardiac asthma),[199] and this gives rise to dyspnea (breathlessness); the heart, in particular the left ventricle, is always enlarged in such cases and the etiological factor is generally obvious. Failure of the right heart chambers, essentially right ventricular failure, is evidenced by an increase in systemic venous pressure revealing itself in engorged neck veins, enlarged liver, and swelling (edema) of the legs. There may be two factors back of congestive heart failure: a chronic process such as high blood pressure or valvular disease on the one hand and on the other an acute factor such as acute coronary thrombosis, the onset of the rapid heart rate of auricular fibrillation, pulmonary embolism, or trauma or strain; the relative responsibility of each of the factors very often may be judged with a fair degree of assurance. Congestive heart failure varies greatly in its prognosis (outlook), depending chiefly on the severity both of the underlying heart disease and of the exciting factor. If the chronic heart trouble is great in degree so that only a slight strain or none at all is needed to cause congestion the prognosis is bad, a matter of a few months to a year or two at most, dependent largely on the treatment. If, however, there is not a great deal of chronic heart disease and the exciting factor is severe but only transient or remediable there may be an astonishingly good recovery with years of survival. On the other hand, even though there be but little chronic heart disease or none at all, the acute process, no matter what—infarction, trauma, or otherwise—may kill quickly.

Functional disorders of the circulation (continued): *Insufficiency of the coronary arteries in maintaining the blood supply of the heart.* Coronary insufficiency is also a serious condition, often rapidly fatal but many times proving to be transient and compatible with complete recovery and survival of the patient for many years. It is most commonly due to chronic coronary artery sclerosis[200] and obstruction with or without acute thrombosis (formation of a plug or clot by coagulation of the blood). Syphilitic aortitis[201] obstructing the mouths of the

[197] *Extensive pulmonary disease:* Extensive pulmonary (lung) disease acts to produce enlargement of the right side of the heart by increasing the blood pressure in the pulmonary circulation.

[198] *Congenital defects:* Congenital defects act in various ways to increase the work of the heart, particularly of the right ventricle.

[199] *Cardiac asthma:* Cardiac asthma is a type of asthmatic breathing set off by acute congestion of the lungs. Most asthma is not cardiac, but bronchial.

[200] *Coronary artery sclerosis:* Coronary artery sclerosis is hardening of the coronary arteries usually with considerable narrowing of the lumen or internal diameter of the vessel.

[201] *Syphilitic aortitis:* Syphilitic aortitis is inflammation of the aorta due to syphilis.

coronary arteries is another much rarer cause. Other etiological factors are very rare. It is not produced by nervousness alone or by the uncomplicated effect of tobacco. Evidence of coronary insufficiency consists of only three criteria, namely:

1. Angina pectoris (paroxysmal chest pain) clearly elicited by careful questioning (or perhaps by testing by exercise or low oxygen—10 per cent inhalation) and not easily simulated by a malingerer;

2. Electrocardiographic abnormalities in characteristic patterns (chiefly changes in *S-T* segments and *T* waves), and auriculoventricular and bundle branch block not ascribable to other causes;[202]

3. Clinical course of acute myocardial infarction. As in the case of myocardial insufficiency with congestive heart failure, so here, also, there may be two factors responsible, one a more or less chronic state of coronary heart disease with or without an actual myocardial infarct or scar, and the other (rare) an acute strain—tachycardia, operation, trauma or emotional stress; such acute strain would not be regarded by scientific investigators as the sole cause although it may serve to bring the patient's condition to light. Precise apportionment of the relative responsibilities of the two factors often requires keen judgment and rich experience but an approximation is more accurate and just than attributing all or none of the claimant's condition to the traumatic episode.[203]

Functional disorders of the cardiovascular system (continued): *Irregularities of the heart beat.* The arrhythmias of the heart should receive our attention next. Although they may often be detected and properly interpreted by clinical examination alone, electrocardiographic

[202] *Other causes:* There are rarely other causes of electrocardiographic changes resembling those due to coronary insufficiency. These other causes are marked digitalis intoxication, severe infectious disease, pericarditis, and moribund conditions.

[203] The law has not been very scientific in respect to apportioning causation as a means of determining the proper damages due; in personal injury cases, governed by tort law, an *all or none* principle of causation is followed: either the defendant's conduct is considered to have been a substantial cause, raising a liability for full damages, or no cause, and the tort feasor is not allowed to mitigate his damages on the theory that the plaintiff, by virtue of prior infirmity, suffered greater injury than would have a person possessed of average health and resistance. Some, but very few, of the Workmen's Compensation statutes provide for apportioning causation. In the main the statutes provide, or courts construe them to mean, that the employer takes the risk of the employee's pre-existing disease or impairment and if one able to work is disabled by an accident which worsens his condition, the disability is compensable. See SMITH, H. W., AND SOLOMON, H. C., *Traumatic Neuroses in Court,* 30 VA. L. REV. 87 (1943); SMITH, H. W., *Relation of Emotions to Injury and Disease: Legal Liability for Psychic Stimuli,* 30 VA. L. REV. 193 (1944); and see in this symposium, HOROVITZ, S., *Fundamental Principles of the Law of Workmen's Compensation,* IND. L. J. (June, 1946).

But in tort cases and compensation cases alike, a disability which existed prior to the accident is not compensable; in tort cases, particularly, it is important to distinguish sharply between such pre-existing disability (non-compensable) and the worsening or aggravation caused by the injury (compensable).

study is desirable and sometimes essential. The electrocardiogram gives a tracing of the heart beat with the frequency clearly charted against time intervals. The vast majority of persons who show cardiac arrhythmia are healthy and have otherwise normal hearts and their disturbances of rhythm are unimportant even though they may be somewhat uncomfortable and a cause for concern. There is no need for worry and little or no need for treatment in such cases. A large percentage of *cardiac neurosis* and of *neurocirculatory asthenia* (see discussion of both, *infra*) seems to have an origin in the fear and discomfort occasioned by arrhythmia of little or no importance. There are, however, a few serious disorders of rhythm.

Premature beats, or extrasystoles, as they are sometimes called, are very common and are to be considered a normal finding in older persons when they do not occur constantly every few beats. Mostly of ventricular origin, they result from an early discharge of stimulus which thus produces a heart beat arising in a part of the heart muscle not directly at either auricular or ventricular pacemaker. The reason for this premature discharge of the cardiac stimulus is unknown in many cases, oftentimes, however, it appears to be caused by tobacco smoking, indigestion, the toxic effect of certain drugs, especially digitalis, by infections, or nervous fatigue; again, it may be caused by heart disease itself, as, for instance, by myocardial infarction[204] and mitral stenosis.[205] It may or may not be felt as palpitation by the person himself. It does no harm. It is shown by the prematurity of a heart beat disturbing otherwise normal rhythm, and it is best seen in the electrocardiogram. The prognosis is excellent. It may occur even as often as every other beat for many years without harm to life or health except as it may induce an anxiety neurosis[206] in the person involved. If there is much discomfort from it, it is often helpful to eliminate any obvious possible cause such as tobacco and to try the effect of quinidine sulphate or bromides or phenobarbital.

Paroxysmal tachycardia of auricular origin is next in order. It consists of spells of rapid racing of the heart at a rate of 140 to 200 per minute, as a rule with sudden onset and offset, and lasting a few seconds to a few hours. Like premature beats, it is of little or no importance and is generally found in persons without evidence of heart disease, but it is usually more disturbing and more likely still to give

[204] *Myocardial infarction:* See note 5 and note 195, *supra.*

[205] *Mitral stenosis:* See note 39 and note 196, *supra.*

[206] *Anxiety neurosis:* Neurosis is a functional disorder of the nervous system not dependent on any discoverable lesion. Anxiety neurosis, by far the most common type seen among soldiers subject to strenuous combat conditions, is characterized by anxious apprehensions and a variety of symptoms which may or may not be associated with any one organ.

rise to neuroses. When superimposed on heart disease itself it may precipitate myocardial or coronary insufficiency and so, in such cases especially, it needs treatment such as can be found outlined in texts on heart disease. It is best identified by electrocardiography which distinguishes it from the much more important *ventricular paroxysmal tachycardia.* In the absence of heart disease it is compatible, even though frequently recurrent, with many years of life and health, in fact it does not shorten life.

More pronounced disorders of *auricular rhythm,* but still possible in the absence of heart disease, are *auricular fibrillation and flutter.* They are the result of a very rapid *circus wave*[207] of excitation in the right auricle coursing irregularly in the case of fibrillation and regularly in the case of flutter, at rates of 200 to 400 per minute. The ventricles are not able to follow the fast beat so initiated by the auricle: in the case of *fibrillation* the ventricles contract absolutely irregularly at the rate of 120 to 160 beats per minute, in the case of *flutter* the ventricles usually contract regularly at a rate close to 150 per minute due to the presence of a 2 to 1 a-v block. (That is, every other impulse arising in the auricle excites a ventricular contraction while every other impulse is blocked.) If the heart is normal to start with, the strain of the tachycardia (rapid heart rate) of such arrhythmia is well borne. But if the heart is seriously diseased, myocardial or coronary insufficiency may be induced, the former much more commonly than the latter; control, that is, reduction, of the ventricular rate by digitalis in such cases usually clears the congestive failure. When a doctor in the clinic, listening with a stethoscope, detects an absolute irregularity of the heart rhythm, this almost invariably means *auricular fibrillation.* Electrocardiography is important for its absolute identification, however, and to diagnose *auricular flutter* with certainty an electrocardiogram is often essential. As a matter of fact, routine electrocardiography sometimes reveals an entirely unsuspected *auricular flutter.* Auricular fibrillation and flutter may be permanent or they may occur paroxysmally for periods of minutes to hours or days. They are most commonly encountered in cases of rheumatic heart disease with mitral stenosis and in cases of thyrotoxicosis.[208] The prognosis in auricular fibrillation and flutter is good

[207] *Circus wave:* The circus wave is a wave of contraction of muscle going round in more or less of a circle, usually at a very rapid rate and found characteristically in the conditions of auricular fibrillation and auricular flutter. Fibrillation is an incoordinate rapid action of the auricles from the circuit of the excitation wave at a very rapid rate with a very irregular pulse at the wrist before treatment is instituted, while flutter is its first cousin with a much more regular action at a slower rate and with regular beating of the heart.

[208] *Thyrotoxicosis:* A disease caused by excessive activity of the thyroid gland, characterized by sweating, weight loss, increased metabolic rate, tremors and sometimes increased prominence of the eyeballs (exophthalmos). See note 82 *supra.*

as to continuation of life, in the absence of structural heart disease or of coronary insufficiency, and even as to comfort if the ventricular rate is controlled adequately by digitalis or if paroxysms are prevented or quickly abolished by use of the drug *quinidine*.

Ventricular paroxysmal tachycardia[209] is serious, nearly always being a sign of grave heart disease (myocardial infarction particularly) or of digitalis intoxication.[210] It requires electrocardiography for diagnosis; otherwise, in its rate and rhythm and duration, it resembles auricular paroxysmal tachycardia. It often leads to *ventricular fibrillation*[211] and death but there are a few benign cases and quinidine can rescue others by abolishing the arrhythmia.

Heart block is of three kinds and of various grades and seriousness. The term refers to blocking of the spread of the impulse or excitation wave from one part of the heart to another. *Sinoauricular block* is the least important and rarest kind, consisting of a pause in the entire heart beat at the pacemaker itself; it is usually not a sign of heart disease. *Auriculoventricular block* varies from a very slight grade of slowing of conduction of the stimulus from the auricles to the ventricles (demonstrated most clearly by the finding of a prolonged *P-R* interval in the electrocardiogram beyond 0.2 second), to dropped beats (ratio of auricular to ventricular contractions varying from say 6 to 5 down to 2 or even 3 to 1), to complete dissociation in which the auricles and ventricles beat independently at their own rates of about 70 to 80 per minute for the auricles and 30 to 40 per minute for the ventricles. The causes of auriculoventricular block are few in number, and two in particular, namely coronary heart disease, acute or chronic, and acute rheumatic heart disease. Among the very rare causes are cardiovascular syphilis, diphtheria, congenital defects, neoplastic (cancerous) infiltration, and trauma. The rheumatic type of block is rarely of high degree; almost all the cases show simply slight prolongation of the *P-R* interval for a few days or weeks. High grade block is almost invariably a sign of a serious degree of coronary heart disease, compatible, however, with years of life and usefulness; the only direct hazard is from syncope (fainting) and death in very few cases in which the ventricles cease to

[209] *Paroxysmal tachycardia:* Paroxysmal tachycardia is a condition marked by attacks of excessively rapid heart action which come on abruptly and terminate just as abruptly. The term "tachycardia" is usually applied to a pulse rate above 130 per minute. "Ventricular" indicates that it is the ventricle which initiates this rapid rate.

[210] *Digitalis intoxication:* Digitalis intoxication is poisoning by excessive amounts of digitalis or a reaction of a sensitive person to usual amounts or even to relatively small dosages.

[211] *Ventricular fibrillation:* A condition of muscular action in which the individual muscle fibers of the ventricle take up their own independent action, producing an incoordinate or vermicular contraction. As a result the heart fails to function and the pulse stops.

contract for sizable fractions of a minute, for example 10 to 20 seconds (Morgagni-Adams-Stokes syndrome). Identification of the block is best made by electrocardiogram which is likely to show other evidence of trouble if the etiological factor is coronary, and nothing else wrong if rheumatism is to blame. Finally, *bundle branch block* is the third type; it is due almost invariably to coronary heart disease which may be silent otherwise and it can be identified only by the electrocardiogram. The prognosis is variable but the outlook for the patient may be good for many years in the absence of other signs or symptoms of trouble. Frequently the discovery of bundle branch block is accidentally made during a routine examination for insurance or for military service.

Functional disorders of the cardiovascular system (continued): *Neurocirculatory asthenia.* The condition labelled *neurocirculatory asthenia* (effort syndrome, soldier's heart) is not heart disease, in fact it is hardly to be designated a disorder of the heart at all, but since cardiac symptoms are a prime feature of the condition, since it is so common, and since it is frequently confused with heart disease, it is necessary to write briefly about it. Neurocirculatory asthenia is a state of poor health characterized by nervousness, easy exhaustion, dyspnea with sighing, palpitation, precordial aches and pains,[212] and a tendency to faint. It is frequently superimposed on or set off by anxiety neurosis[213] and may complicate heart disease. As an acute state it may temporarily complicate convalescence from an infection or follow great or prolonged physical or nervous strain. Many individuals born with hypersensitive nervous and circulatory systems are prone to develop neurocirculatory asthenia under any strain, nervous, physical, infectious, or traumatic; they may suffer from it as a chronic condition, often inherited.[214]

Functional disorders of the cardiovascular system: cardiac neuroses. Cardiac neuroses have become very common based partly on widespread publicity about the increasing incidence of heart disease and partly on exaggeration of the significance of cardiac symptoms and signs[215] in the

[212] *Precordial aches and pains:* Aches and pains over the *precordium,* roughly the region over the heart and stomach.

[213] *Anxiety neurosis:* See note 206, *supra.*

[214] A considerable number of cases of neurocirculatory asthenia occurred in World War I, with many resultant claims under War Risk Insurance Policies. National Life Insurance issued to men in service in World War II insured against death only but the services have had to consider the line of duty status of such disabilities for pension purposes. In the main the tendency is to deny service connection, and to refuse line of duty status on the ground that the condition existed prior to enlistment unless the symptoms first appeared following strenuous combat.

For cases of *traumatic neurosis* with symptoms centered on the heart, see Smith, H. W., and Solomon, H. C., *loc. op. cit. supra,* note 203.

[215] *Symptoms and signs: Symptoms* are subjective states, such as pain, felt by the patient and described by him; *signs* are the objective evidences of disease observable by acute observation, as, for instance, a rash or a swollen abdomen.

minds of the victims of such symptoms and signs. Trauma or other strain in a nervously sensitive individual is very likely to cause such a neurosis, especially if there has been any injury in the region of the heart or if cardiac arrhythmia or neurocirculatory asthenia has resulted or been made worse. The prognosis as to recovery depends partly on the intelligence of the patient, partly on the clearing up of factors of strain and of the symptoms therefrom, but especially on adequate treatment which includes sympathetic reassurance, rest when needed, and reeducation and rehabilitation. Most important of all, however, is prevention which is in great degree possible, if at the very onset of heart trouble, "organic" or "functional," the physician spends an adequate amount of time making a careful examination and giving a full explanation to the patient himself.

Pervasive Medicolegal Aspects of Cardiac Conditions

This section is by no means intended to serve as a complete recapitulation of what has gone before, but rather as a quick drawing together of cardinal medicolegal principles which should be borne in mind when the lawyer has contact with claims or litigation concerning the heart.

1. *Is plaintiff's pre-existing heart disturbance or disease compatible with his professed ignorance of his condition?* An insurance company seeks to cancel a policy on the ground that the assured, in applying for life insurance, fraudulently concealed his past heart disease. Or a defendant in a personal injury action seeks to discredit the plaintiff's claim that if he had heart trouble before the accident occurred, he did not know it.

The fact is that valvular defects, coronary disease, hypertension (high blood pressure), and disorders of rhythm may exist without being evident to casual observers or known by the subject himself. Careful medical examination, however, clearly reveals them, except in the case of minor or transient effects. This is an important reason why *careful, annual physical examinations should be made of all industrial workers* exposed to physical exertion, strain or accidental injury and it is an argument for a periodic health census and examination of the whole populace. In the absence of such systematic and adequate medical examinations, an individual who is a bad risk may be granted life insurance; a person perfectly capable of limited exertion may be put to work at tasks so strenuous that a cardiac disaster is precipitated; an individual may honestly ascribe his entire cardiac impairment to a traumatic episode, physical or psychic, when in fact his heart was badly impaired beforehand and his condition was only aggravated by the accident.

Many unimportant cardiac lesions or disorders require no particular regulation of the subject's life; others diminish the cardiac reserve somewhat while still permitting useful employment at industrial tasks which are not strenuous, and still others circumscribe more closely the permissible types of employment and require concurrent precautions and treatment.

2. *General principles of cardiac injury.* Anyone who has occasion to consider alleged cardiac injury may be helped by the following simple rules which are well established medically, namely:

a. *First, one must consider whether the traumatic stimulus acted upon the claimant, whether it was adequate to produce an injury of the sort alleged, and whether, from all the evidence, it probably did do so. In considering this question one must know whether the claimant contends that the traumatic stimulus injured a previously healthy heart or merely aggravated a pre-existing cardiac impairment or disease.*

b. *Other things being equal, the more severe the injury to the thorax (chest), whether penetrating or not, the more likely is the heart to be affected.*

c. *The more diseased the heart is, the more easily it is injured by trauma, and within limits, the less violent will be the stimulus required to precipitate injury.*

d. *The more nervously sensitive the subject, the more numerous and more severe will be his symptoms following trauma, whether the heart is damaged or not.*

e. *The signs of cardiac injury may be readily evident from quick physical examination, or they may be so obscure that all recognized methods of cardiac examination, including electrocardiography and roentgen-ray study, must be employed.*

f. *Proof of causal connection between the alleged traumatic stimuli and the alleged cardiovascular injury requires careful consideration of what is scientifically possible and what is medically probable in the particular case.*

g. *The patient's prognosis (outlook) following cardiac injury depends upon several factors, namely:*

(1) *The nature and degree of the cardiac injury;*

(2) *The nature and degree of pre-existing heart disease or impairment;*

(3) *The nature and degree of non-cardiac complications;*

or impairments which result from the trauma or preceded it; and

(4) *The nature and sufficiency of treatment, emergency and convalescent.*

Let us now consider each of these principles a little more fully, using them as focal points around which we may better organize discussion for the benefit of the lawyer.

3. *General principles of cardiac injury:* a. *Did the traumatic stimulus act upon the claimant? Was it adequate to produce an injury of the sort alleged? Considering all the evidence did it probably do so?* To answer whether or not the traumatic stimulus actually existed and whether or not it operated injuriously upon the claimant requires both non-medical and medical evidence to be marshalled and appraised with extreme care in the light of all the scientific knowledge we possess concerning injury of the cardiovascular system.

We have seen that the heart is a strong and tough muscular pump, able to carry on a tremendous amount of work over many years despite defects in its structure or function. Most of the diseases that affect it are in no very particular way induced by the stress and strain of occupations or by injuries. This remark applies to the causation of the different kinds of heart disease, the most common of which are rheumatic, hypertensive, and coronary; the less common types including congenital, bacterial endocarditis, syphilitic, thyrotoxic, and pulmonary are, likewise, in the main, unrelated to the hazards of occupation or to traumatic injury. There are exceptions but they are few in number and are mostly unimportant. Such exceptions include the following:

a. The infrequent precipitation of acute rheumatic infection, which can involve the heart, by some injury or unusual exposure;

b. The production of the *chronic cor pulmonale* (enlarged right ventricle, etc., from lung disease or conditions increasing resistance in the pulmonary circulation), by silicosis resulting from the inhalation of dust containing silica;

c. The rather infrequent, severe, direct and indirect traumatic lesions of the heart itself; and

d. The production of pulmonary emboli (blockage of pulmonary artery or its branches by blood clots, etc., transported in the blood) with secondary *acute cor pulmonale.*

The aggravation of pre-existing heart disease by industrial strain and accidents and by traumatic stimuli is, however, very common, as, for example, when a person with rheumatic mitral stenosis develops the

complication of auricular fibrillation with rapid heart rate which may induce pulmonary (lung) congestion or failure of the right heart chambers; or the individual with enlargement of the left ventricle of the heart secondary to high blood pressure develops, under special strain, acute dilatation of the left ventricle with edema (watery congestion) of the lungs; or a patient with old or new myocardial infarction (death of a limited area of heart muscle from deprivation of its blood supply) secondary to coronary thrombosis (blockage of a coronary artery with consequent reduction of the blood supply of the heart muscle) overstrains his already damaged left ventricle and develops heart failure; or a man with angina pectoris (sudden excruciating heart pain felt over the chest and sometimes radiating to the arms) has a fatal attack under special provocation; or a victim of cardiovascular syphilis with an aortic aneurysm (ballooning out of the weakened aortic wall) suffers strain which ruptures that aneurysm.

Thus, the very first question to be answered is this: is the case one of alleged injury to a previously healthy heart or one of aggravation of a pre-existing cardiac impairment or disease? This will affect the measure of damages recoverable in a tort action for personal injuries; it will not affect the amount of benefits in a workmen's compensation case except in those few states whose laws provide for apportionment of disability between accidental injury and pre-existing disease; but it will materially affect the question as to whether the nature and degree of the traumatic stimulus have been sufficient to affect the claimant's heart injuriously.[216] The plaintiff or claimant who asserts that the impairment to his heart is due entirely to the traumatic episode thus has a very difficult burden of proof to discharge; if he claims aggravation of a pre-existing cardiac impairment or disease, the medically known physiological mechanisms by which such worsening of his heart condition may occur come into play, and a less severe provoking injury on the whole would be required to establish a cause-effect relationship recognizable scientifically. Both parties may thus have an interest in proving pre-existing cardiac impairment or disease: the plaintiff, to enable him to meet an otherwise difficult burden of proof in establishing causal connection; the defendant, to minimize damages or to lay a foundation for rebuttal evidence that the plaintiff's cardiac disaster was due solely to his heart disease independent of the traumatic stimulus sought to be incriminated. The legal reader should understand that

[216] It appears from a review of cardiac litigation that the great majority of workmen's compensation cases are in fact disabilities due to aggravation of pre-existing disease. The commissions first accepted the extraordinary exertion as the equivalent of an accident at work; it appears that some are now going further and in effect are holding that an unintended, unanticipated cardiac injury resulting from any exertion at work is a compensable accident.

adequate examination by a heart specialist, utilizing all necessary diagnostic aids, will ordinarily permit him to establish the type of heart disease the plaintiff has, if any, and oftentimes, by reasoning from the degree of progression, to estimate how long the disease has existed.[217] Lawyers will think of other sources of evidence for proving pre-existing heart disease: utterances of the plaintiff revealing a prior awareness, medical records of past examinations and treatment, previous hospital admissions and the like.

From the standpoint of proving causal connection and the proper measure of damages, it is important that the defendant, by proper investigations, depositions and pleadings, compel the plaintiff to stand on one or the other of the following theories concerning his alleged injury, namely:

a. Cardiac disease or impairment directly caused by a traumatic stimulus;

b. Pre-existing cardiac disease or impairment aggravated by a traumatic stimulus; or

c. Cardiac symptoms caused by a traumatic stimulus without any ascertainable injury to the cardiovascular system (neurocirculatory asthenia or cardiac neurosis).

Some aid is gained by classifying traumatic stimuli as *direct* (physical impact or strain, etc., applied directly to the heart) and *indirect* (injury to a remote part of the body, causing surgical shock and cardiovascular collapse; emboli passing in the blood from a remote part of the body, as for instance an injured leg, etc., psychic stimuli, etc.). One may also classify traumatic stimuli in the order of ease in demonstrating causal connection with the alleged injury, progressing from the more obvious types to the more obscure, as follows:

a. Most obvious of all is the penetrating wound (knife stab, bullet wound, etc.);

b. Definite, but not so obvious, is contusion (bruising of the heart) by a blow on the chest, or by a crushing injury. This mechanism of injury includes direct injury from a fall against a hard object, or the ground, or the surface of water.

c. Less obvious still is the connection between indirect jarring, or of a strain from a fall or sudden effort, as, for instance, the effort exerted to lift a heavy weight or to prevent slipping.

d. More obscure still are injuries to the heart from roentgen-ray

[217] For this reason it has seemed superfluous to enumerate in this study the findings necessary to support the diagnosis of each particular type of heart disease; furthermore, these diagnostic criteria may readily be found in text books. See WHITE, HEART DISEASE, 3rd ed. (1944); *Nomenclature and Criteria for Diagnoses of Diseases of the Heart*, 4th ed. (1940); LEVINE, CLINICAL HEART DISEASE, 3rd ed. (1945).

exposure,[218] from various poisons and gases (carbon monoxide: CO; and hydrogen sulphide: H_2S) and from electric currents;[219] lasting injuries to the heart from these sources have not been proved in man, and whatever damage results is probably only temporary, disappearing with the patient's rapid or gradual recovery from the immediate effects of the trauma.

e. Finally, the most difficult claims of causal connection to appraise may be disorders or injury allegedly caused by nervous strain, fatigue, and excitement, fear and psychic stimuli generally.[220]

4. *General principles of cardiac injury: The more severe the injury to the thorax (chest), whether penetrating or not, the more likely is the heart to be affected.* The principle stated is self-evident; but certain subsidiary facts deserve mention. One is that any chamber of the heart may be ruptured without a penetrating injury.[221] The heart chamber is distended with blood at the end of diastole (resting phase) or the beginning of systole (pumping phase). At this time, sudden and extreme pressure applied to the heart chamber is far more likely to rupture it, whether it be healthy or diseased, than when it is partially or nearly empty (in early diastole or at the end of systole). In general, compressive forces applied antero-posteriorly to the chest are of more serious consequence than those applied at either side. This is because the antero-posterior (chest-back) type of compression squeezes the heart between two bony structures, the rib cage and the backbone, and thus interferes with filling and functioning of the heart to a greater extent.[222]

[218] See, in this symposium, DUNLAP, C. E., *Medicolegal Aspects of Injuries from Excessive Irradiation with X-ray and Radium*, OCCUPATIONAL MEDICINE (April or May, 1946).

[219] See, in this symposium, HYSLOP, GEORGE H., *Medicolegal Aspects of Injuries by Electrical Shock, with Special Reference to the Nervous System*, TENN. L. R. (April, 1946); OCCUPATIONAL MEDICINE (April or May, 1946).

[220] It seems, in general, that such stimuli, if intense enough, may cause transient elevation of the blood pressure, a variable degree of spasm or constriction of the coronary arteries, an arrhythmia, or an attack of angina pectoris. Death or injury through the effect of psychic stimuli on the cardiovascular system should be explicable in terms of some such known mechanism, and all the clinical and post-mortem evidence should attest its probable occurrence while ruling out any other cause of death.

[221] In 105 cases involving rupture of a heart chamber, the patient was run over by a train, automobile or wagon in 38; in 22, he was crushed between two objects; in 17, the injury was from a direct blow over the chest, as with a fist or a club; in 12, the patient was struck by a falling object; in 8, the patient's chest was kicked by a horse; in 3, there was injury by an explosion; in 2, a bullet struck the sternum (breastbone); in 1, the injury was due to a fall; in 1, playmates jumped upon the patient and in one the patient was engulfed in a sandbank. The writers summarizing the series considered how many cases might have been saved by surgery; there were 30 of these, who lived from forty-five minutes to an hour after the trauma, and whose injuries were not too extensive to be repaired by prompt suture (surgical sewing). BRIGHT, E. F., AND BECK, C. S., *Non-penetrating Wounds of the Heart*, 10 AM. HEART J. 293 (1935).

[222] BRIGHT AND BECK, *ibid.*, showed, in analyzing 152 cases of death from rupture of the heart, that three different mechanisms may cause rupture of a cardiac

It is important, also, to inquire as to the location of the traumatic injury: blows directly over the cardiac area are more apt to cause injury to the heart than blows to some other part of the thorax except when jagged ends of broken ribs contuse (bruise) or lacerate (tear) the heart, or penetrate one of its chambers.

5. *General principles of cardiac injury: The more diseased the heart is, the more easily it is injured by trauma, and within limits, the less violent will be the stimulus required to precipitate injury.*

If a person dies suddenly following a modest accident or traumatic episode, and with no history of pre-existing heart disease, there is a strong probability that his death was not due to cardiac injury and a postmortem examination is distinctly desirable, and oftentimes essential, to discover the cause of his demise. In *very rare cases,* it seems that substantial injury may cause rupture of healthy heart valves.[223] Of the 38 cases of valvular rupture (without rupture of the ventricular wall) reported by Barié,[224] 19 involved the aortic valve, 16 the mitral, and 3 the tricuspid. Rupture of the aortic valve, in several cases weakened by pre-existing disease, resulted from such episodes as the following: A 33-year-old man, while lifting an enormous piece of coal; a 44-year-old man, after pushing a heavy cart uphill; a blacksmith, after a violent effort in swinging his hammer; a 33-year-old sailor, while climbing the shrouds on his ship; a 45-year-old ditch digger, who fell 15 feet to hard ground; a sailor, who fell to the deck from aloft; a 44-year-old miner who was struck in the chest by a wagon pole; and a 53-year-old wagoner who was kicked in the chest by a horse. The effect of valve rupture on the heart is usually a serious one, but it is rarely, if ever, immediately fatal.[225] Cardiac enlargement and congestive failure usually follow in a period of months or years; if, however, the valve rupture occurs in a heart already impaired by disease, it may precipitate auricular fibrillation and congestive failure quite rapidly.

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chamber, namely: 1. Compression of the heart between the sternum (breast bone) and vertebrae (backbone), similar to compression of a toy balloon in one's hand. 2. Contusion (bruising) of the heart, with subsequent softening of the area of contusion. 3. Increase of the intracardiac pressure by forces applied directly to the heart or as a result of indirect forces generated by sudden compression of the lungs or abdomen.

[223] How rarely it occurs is shown by the fact that Howard, in reviewing the medical literature in 1928, was able to collect only 112 authentic cases of rupture of *healthy or diseased* aortic valves. HOWARD, C. P., *Aortic Insufficiency Due to Rupture by Strain of a Normal Aortic Valve,* 19 CANAD. MED. ASSN. J. 12 (1928).

[224] BARIÉ, E., *Recherches cliniques et experimentales sur les ruptures valvulaires du coeur,* 1 REV. MED. DE PARIS, 132, 309, 482 (1881).

[225] The murmurs, systolic as well as diastolic, produced by ruptured valves often have an extraordinary whirring or musical character. The examiner must take care to distinguish murmurs due to independent causes, as, for instance, pre-existing valvular disease.

Quite aside from rupture of a valve, trauma may precipitate myocardial (heart muscle) insufficiency, even to the degree of extreme congestive failure, in an already damaged heart, whatever the cause of the impairment or the character of the pre-existing structural defects. Such myocardial insufficiency, if caused by trauma, should, with one exception,[226] begin within a few hours after the trauma; it may be only slight at first, and require days or weeks to result in a high degree of congestive failure, or it may produce a maximal degree of congestion within a few hours, even terminating in death.[227]

We have discussed coronary occlusion and thrombosis earlier in this paper but not enough has been said about coronary insufficiency (insufficient blood supply carried to the heart by the coronary arteries) manifested only by angina pectoris or sudden death without other adequate cause. The occurrence of *angina pectoris*—pain in the heart region and substernal oppression (with or without radiation to the arms), induced usually by effort, lasting a few minutes, and relieved by rest or nitrites—ordinarily may be explained in one of three ways:

a. Coronary disease (arteriosclerosis) may have reduced the blood supply to the heart muscle to such an extent that exertion beyond a certain point raises the metabolic demands of the myocardium (heart muscle) above the maximal level of the coronary blood supply.

b. An important portion of the coronary blood supply may suddenly be withdrawn, as the result of vasospasm—spasm of coronary vessels—(coronary vascular crisis), induced reflexly by effort or excitement, with or without much coronary disease.

c. Both causes may operate together. Our knowledge concerning the relative frequency of one or the other of these factors in angina pectoris is practically *nil* at the present day.

Trauma may precipitate an individual attack of angina pectoris, which may in turn, in rare cases, end quickly in death; it apparently may initiate the symptoms which will recur at intervals in the future; it may simply induce an attack similar to, or more or less severe than, other attacks in the past; it may precipitate coronary thrombosis in a patient who has been having attacks of angina pectoris; or finally, it may have

[226] The exception is found in a group of cases in which some structural damage or functional derangement, caused by the cardiac trauma, acts only gradually to tire the heart. Examples of such are cases with rupture of a valve, and with prolonged paroxysmal tachycardia, auricular flutter or auricular fibrillation. Days, weeks, months, or even years may elapse between the time of the trauma and the onset of the myocardial insufficiency and congestive failure in these cases. In such cases, great care must be taken to see whether independent causes have entered the picture; the plaintiff's medical and social history must be scrutinized closely.

[227] As in the case of fulminating pulmonary edema (massive watery congestion of the lung), due to acute failure of the left ventricle.

no appreciable effect at all in a case of angina pectoris, whether of recent origin or of long standing. Whether the trauma acts directly, through its contusive (bruising) effect on the coronaries and heart (as Schlomka's experimental work suggests) or whether it acts reflexly through nervous excitement, we cannot say. The time interval between the trauma and the resultant angina pectoris is only a matter of minutes at the most.

The strain of hard physical or mental work, or intense psychic stimuli, may precipitate angina pectoris but they are not known to have any causal relation in producing the underlying atherosclerosis.

Angina pectoris is sometimes curable by rest; sometimes it stops spontaneously, but often it remains as a hazard, recurring under sufficient provocation. It rarely leads at once to early death.

Sudden death as the result of cardiac trauma may be due to angina pectoris or to the condition of coronary insufficiency it symbolizes. In the absence of other demonstrable cause by postmortem examination, it may be so diagnosed if the subject complained of the characteristic pain during or immediately after the traumatic experience and just before his death, or if he was known to have been the victim of attacks of angina pectoris in the past.[228] Just how death comes in most cases of angina pectoris we do not know as yet—whether by overwhelming depression of the cardiac pacemakers, with immediate and total cardiac standstill, or by the abrupt onset of ventricular fibrillation (irregular, incoordinate contraction of the ventricles), or by some other mechanism.

6. *General principles of cardiac injury: The more nervously sensitive the subject, the more numerous and more severe will be his symptoms following trauma, whether the heart is damaged or not.*

It is important to determine whether the plaintiff's symptoms are referable to an injury or disturbance of the heart or whether they are in the nature of psychological reactions without any demonstrable organic basis (traumatic neurosis). In the latter case, the more scientific approach is to consider whether the same stimulus would have affected an average person in a similar way; if not, development of a neurosis in response to the insubstantial stimulus should be regarded as a mere aggravation of a pre-existing neurotic diathesis and the dam-

[228] In such cases, in the absence of any extraordinary accident or strain, or where the time interval between the anginal attack and subsequent death is two or three weeks, without strong evidence of a continuous cardiac illness, courts are apt to deny compensation, as in Black Forest Fox Ranch v. Garrett (Colo. Sup. Ct.) 134 P. (2d) 332 (1943), and Duncan v. Weidman (Nebr. Sup. Ct.) 11 N. W. (2d) 537 (1943). But where excitement of detecting a fire causes a prompt and fatal attack of angina pectoris, compensation is allowed, even in those states such as Michigan which refuse to permit recovery of damages in tort cases (Nelson v. Crawford, 122 Mich. 466, 81 N. W. 335 (1899)) for injuries caused by fright or psychic stimuli without contemporaneous impact. Schroetke v. Jackson-Church Co., 193 Mich. 616, 160 N. W. 383 (1916).

ages awarded should be modest rather than large.[229] The same reasoning applies to *neurocirculatory asthenia,* a symptom complex previously described in this paper. It involves functional disturbances which may, like cardiac neurosis, be responsible for all the symptoms and many of the signs in persons who may or may not have any heart disease or who have been under special strain. Some individuals are born with a tendency to neurocirculatory asthenia. They should be recognized early and so labelled, prior to aggravation of symptoms by trivial causes during the passage of the years.

7. General principles of cardiac injury: The signs of cardiac injury may be readily evident from quick physical examination, or they may be so obscure that all recognized methods of cardiac examination, including electrocardiography and roentgen-ray study, must be employed.

8. Proof of causal connection between the alleged traumatic stimuli and the alleged cardiovascular injury requires careful consideration of what is scientifically possible and what is medically probable in the particular case.

These two principles may be considered together. The fact and cause of cardiac injury may be clear where the stimulus is intensely traumatic, or the individual was peculiarly susceptible to it as a result of demonstrable pre-existing impairment of the heart, and where prompt pain or signs of cardiovascular damage follow within the characteristic time intervals after the accident. We have seen that the time intervals and the clinical progression consistent with a claim of traumatic injury to the cardiovascular system depends upon the nature of the damage done and whether it is superimposed upon a pre-existing impairment of the heart. During the last few years, it has become increasingly evident that slight and even serious cardiac damage may occur after injury, with little or no immediate cardiac symptoms to indicate its presence. Whenever a workman collapses after unusual exertion or has sustained blows to his chest, or complains of angina pectoris or chest pain suggestive of a coronary attack, he should be put to bed and examined thoroughly. This should include careful examination of the heart by inspection, palpation, percussion, auscultation, X-ray, and electrocardiography. Electrocardiograms made at regular intervals (serial electrocardiograms) may provide evidence of traumatic injury when other forms of examination fail to disclose it and they may also localize the exact area of the heart involved.[230] For that reason, interval electro-

[229] See SMITH, H. W., AND SOLOMON, H. C., *Traumatic Neuroses in Court,* 30 VA. L. REV. 87 (1943); 21 ANNALS OF INT. MED. 367 (1944).

[230] When a chest injury occurs in a person in the coronary age group, it may not be such an easy matter to rule out pre-existing coronary disease, if no electrocardiogram was taken previously, except when surgical exposure reveals the

cardiograms should be made routinely in all such cases.[231] In obscure cases which end in death, postmortem examination may well be the only means of reaching a scientific diagnosis.

9. *Pervasive medicolegal aspects of cardiac conditions: Conclusion.* It is not necessary to comment on the several factors which determine the patient's prognosis following cardiac injury (enumerated in 2g. *supra*) as they are self-evident. The attorney will see that the residual disability or final outcome may be long delayed in developing and he should therefore exercise caution in the making of compromise settlements. He should proceed on the basis of thoroughgoing medical study and advice, utilizing a heart specialist (cardiologist) if one is available. Lastly, the defendant's attorney, in personal injury actions not charging heart injury, will do well to have the plaintiff's main organ systems examined, for the reason that pre-existing disease, whether of the heart or of other organs, may account for a good part of the symptoms of disability imputed to trauma.

Once courts and lawyers gain a knowledge of basic facts concerning heart disease and cardiac injury, the risks of mistake or imposition will be lessened. If the present paper helps to promote such an understanding, the writers will be content.

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nature of the injury. However, when characteristic acute changes appear in the electrocardiogram (i.e., inversion of T_1 and T_2 or T_3 indicating coronary insufficiency, or inversion of all T waves indicating pericardial disease) directly following an injury, and are found to progress and later to disappear, this constitutes evidence of myocardial or pericardial damage induced by trauma.

[231] See SMITH, H. W., AND RISEMAN, J. E. F., *Applied Use of the Electrocardiogram in Legal Proceedings*, 15 ROCKY MT. L. REV. 251 (1943); also, RISEMAN, J. E. F., AND SMITH, H. W., *Some Legal Aspects of Heart Disease and the Electrocardiogram*, 19 ANN. INT. MED. 81 (1943).